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# Sudarshan Kriya Intervention and Rise of Protective Prostaglandins in Advanced-Stage Breast Cancer: Double-Blind, Randomized, Controlled Trial

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***Abstract***

***Background:*** *Breast cancer patients in advanced stages of cancer are managed by palliative care. This study reports important prostaglandin changes observed with added intervention of Sudarshan Kriya in their ongoing palliative care. Level of Prostaglandin $A_2$ ($PGA_2$) indicates tumor growth suppression; its level rises if person's general condition is good, physically fit and active while $PGE_2$ and $D_2$ are considered pro-inflammatory. Quantitative assessment of Prostaglandins was done pre and post intervention of Sudarshan Kriya Pranayam (SKP) among 150 stage 4 breast cancer females above 50 years age.* ***Methods:*** *The study conducted in pain and palliative clinic of AIIMS Delhi patients after obtaining informed consent to enroll in standard versus added intervention of SKP. Patient data were kept coded by one team and three other teams worked in double-blinded manner on coded data since randomization/ allocation till intervention and testing using separate teams for opening envelopes for arm allocation and imparting care/ intervention and testing markers. Consenting participants (n = 147) randomized, received standard care (n = 69) and standard care plus SKP (n = 78). SKP arm patients learned Sudarshan Kriya in level one course of Art of Living – 3 hours per day for 4 days. Pre and post intervention blood samples collected at baseline-day 1, and at 3rd month post enrollment/ intervention.* ***Results:*** *A significant difference (rise) in blood prostaglandin levels of $PGA_2$ at 3 month's sample in SKP intervention observed – $PGA_2$ (mean 549.2 ng/ml) versus mean 341.2 ng/ml of control arm ($P \leq 0.002$). $PGD_2$ and $E_2$ levels, however, showed no difference with baseline, 3rd month's samples. $PGD_2$ and $E_2$ both changed in intervention as well as in control arm from its baseline mean, differences are not significant unlike $PGA_2$.* ***Conclusion:*** *Added intervention of SKP during palliative care boosts wellbeing among advanced stage breast cancer patients.*

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**Keywords:** Breast cancer, cancer, inflammation, prostaglandin, Sudarshan Kriya Pranayam (SKP)

## INTRODUCTION

Late detection of female breast cancer leads to high prevalence (28% cases) of advanced stage breast cancer in India [1]. Requirement of palliative and supportive care, therefore, is huge among advanced stage breast cancer patients [2]. This study was conducted using AYUSH Ministry, Government of India funding during 2013–15 [3]. The data analysis and presentation were done for pain and other biochemical parameters during 2015. However, final data related to prostaglandin estimation could not be published for lack of interpretation, availability of staff for data analysis and interpretation on gathered information, published literature and clue on how to define the observations on prostaglandin markers. As prostaglandins are majorly considered pro-inflammatory and the purpose of measuring prostaglandin was to test

level of inflammation among the participants taking treatment and added intervention. Contradictory findings of rising prostaglandins with SKP intervention were difficult to define logically until recent research opened gateways on various modes of action and roles of prostaglandins in causation of inflammation and cancer. A few recently published papers clarified roles of prostaglandins in cancer causations and revealed that not all prostaglandins are pro-inflammatory and some show anti-cancer activities too. Hence, the findings of the study become crucial to help advanced stage cancer patients.

Varieties of inflammatory markers are used to observe cancer progression and prognosis [4, 5]. Among these markers, prostaglandins (PGs), a family of biologically potent lipids from membrane phospholipids, comprised of a $C_{20}$-unsaturated fatty acid containing a cyclopentane ring and play important role in cancer [6]. Increase PGs in inflammation, stress, oxidative injury have been postulated to be important factors in pathogenesis of cancer [7]. Role of cell chemistry of prostaglandins (PG) function in both the promotion and resolution of inflammation is well published [8, 9]. Low concentrations of $PGE_2$ enhance platelet aggregation, whereas high $PGE_2$ levels inhibit aggregation. The mechanism for this dual action of $PGE_2$ is not clear [10]. Not all but some of PG specially $PGE_2$, $PGD_2$ has been established to play role in cancer cell proliferation [11, 12]. Number of published studies suggest that $PGE_2$ increases tumor growth and invasion, reduces apoptosis, increases metastasis and angiogenesis, and suppresses antitumor immunity [13, 14]. However, Prostaglandin $A_2$ ($PGA_2$) recently found to suppress tumor growth *in vivo*, is potently anti proliferative *in vitro* in a mammalian stress response study [15]. Most of the PG-inhibitors (aspirin, ibuprofen, indomethacin, piroxicam, sulindac) or commonly called NSAIDs (nonsteroidal anti-inflammatory drugs) significantly inhibit cancer development and cancer progression by inhibiting $PGE_2$ – a nonselective COX inhibitor as well as selective COX-2 inhibitor [16–18]. Thus, various ongoing research studies identifying approaches to antagonizing COX/PG signaling to use them for cancer prevention and treatment, with a particular focus on $PGE_2$ regulation and signaling, because $PGE_2$ is a key pro-tumorigenic prostanoid [19]. Despite conflicting studies, broadly Prostaglandin $D_2$ ($PGD_2$ & $A_2$) seems to hinder tumor progression, while prostaglandin $E_2$ ($PGE_2$) seem to provide greater tumor progression and aggressiveness [20]. Some studies show $PGE_2$ and $D_2$ causing rise in cAMP, while $PGA_2$ causes fall in cAMP. Contradictory reports show $PGD_2$ have association with inflammatory and atopic conditions, although it might exert an array of immunologically relevant anti-inflammatory functions as well in other published papers [21, 22]. $PGA_2$, however, is now undoubtedly reported to inhibit cancer [23–26].

Mental stress related studies have also found connection between circulating inflammatory markers like prostaglandins and bradykinins [27]. Sudarshan Kriya Pranayam (SKP) Meditation-Yoga has been reported to act via hypothalamus-pituitary-adrenal axis to alter cortisol level [28] and parasympathetic vagal tone to reduce oxidative stress and inflammation and affect mental stress [29]. To do quantitative assessment of prostaglandin levels among the stage 4 breast cancer patients, the blood samples were collected at baseline (at the time of enrollment), and at 3rd months post intervention to see if intervention could bring any change in inflammatory biomarkers with and without SKP intervention.

## METHODS

This double-blind randomized intervention trial was done with ethical approvals from Institutional Ethical committee, AIIMS, Delhi. After informed consent, 187 participants were screened and 38 were excluded for the reasons of non-consenting (n = 5), not having Karnofsky score above 70 (n = 13), were considered for more chemotherapy/radiotherapy (n = 14), age was above 60 (n = 4) or below 35 (n = 2).

Women suffering from invasive ductal carcinoma breast cancer stage IIb to III and IV, have received or completed standard treatment of cancer by radiotherapy, chemotherapy, and surgery and age ranging from 35 years to 60 years, Karnofsky score above 70 were considered fulfilling inclusion criteria. Out of 185 participants screened exclusion criteria applied on 38. 147 patients were allotted unique id of 8-digit number and randomized to receive standard care (n = 69) versus standard plus intervention with SKP (n = 78).

Standard methods of double-blinded randomization were applied by making 5 teams, one for screening recruitment and generating envelopes containing random numbers, allocation, and giving a code number to participant as unique id. Second team treated patients and implemented interventions,

3rd team did documentation and sample collection, and the 4th team did lab work on coded samples received from both arm's cases. 5th team did data analysis and submitted analysis reports to 1st team which finally decoded test reports to make final report and wrote interpretations. Those who decoded codes and those who did analysis on data were separate teams and were not aware of allocation status of the participants. Hence blinding was ensured at each step of study implementation. Samples of 5 ml venous blood fasting in morning were collected to estimate prostaglandin levels prostaglandin $PGA_2$, $PGD_2$, $PGE_2$ along with other parameters of stress.

Table 1 shows the methods used for biochemical analysis: Prostaglandins testing: LC-MS/MS Parameters: Column: Hypersil Gold (50 x 2.1 mm, 1.9 µm), Flow rate: 0.5 ml/min, Run Time: 5 min, Mobile phase: Isocratic [95:5, ACN: $H_2O$], System: Thermo detector: PDA, Software used: Chromquest Ver. 4.1, Source gas Parameters & Values are given in Table 2. Figure 1 shows procedure for standard and sample preparation.

**Table 1.** Methods used for biochemical analysis prostaglandins testing: LC-MS/MS.

| Compound | Q1 | Q3 | Declustering potential (V) | Electrical potential (V) | Collision energy (V) | Collision exit potential (V) | Time (msec) |
|---|---|---|---|---|---|---|---|
| Prostaglandin A2 | 333.4 | 113.3 | –115 | –10 | –40 | –7 | 100 |
| Prostaglandin D2 | 351.2 | 189.3 | –70 | –10 | –30 | –5 | 100 |
| Prostaglandin E2 | 353.3 | 235.1 | –70 | –10 | –25 | –15 | 100 |
| Probenecid as IS | 283.91 | 240 | –115 | –10 | –30 | –15 | 100 |

**Table 2.** Source gas parameters & values.

| Source gas parameters | Values |
|---|---|
| Curtain gas (CUR) | 20 psi |
| Ion Source (IS) | –4500V |
| CAD gas | 8 psi |
| Temperature | 400°C |
| GS1 | 50 psi |
| GS2 | 50 psi |

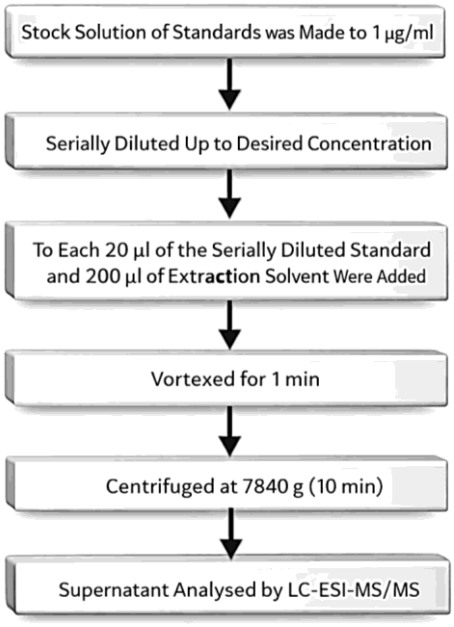



**Figure 1.** Flow diagram of procedure for standard and sample preparation.

## LC-MS/MS PEAKS OF BLANK AND INTERNAL STANDARD PROBENECID

Figure 2 illustrates the LC-MS/MS chromatographic peaks obtained from the blank sample and patient samples, demonstrating the absence of interfering peaks in the blank and clear detection in the

patient samples. Figure 3 presents the standardization of the analytical method, showing the LC-MS/MS peaks of the standard solution prepared at a concentration of 100 ng/mL, confirming the accuracy and reliability of the method.

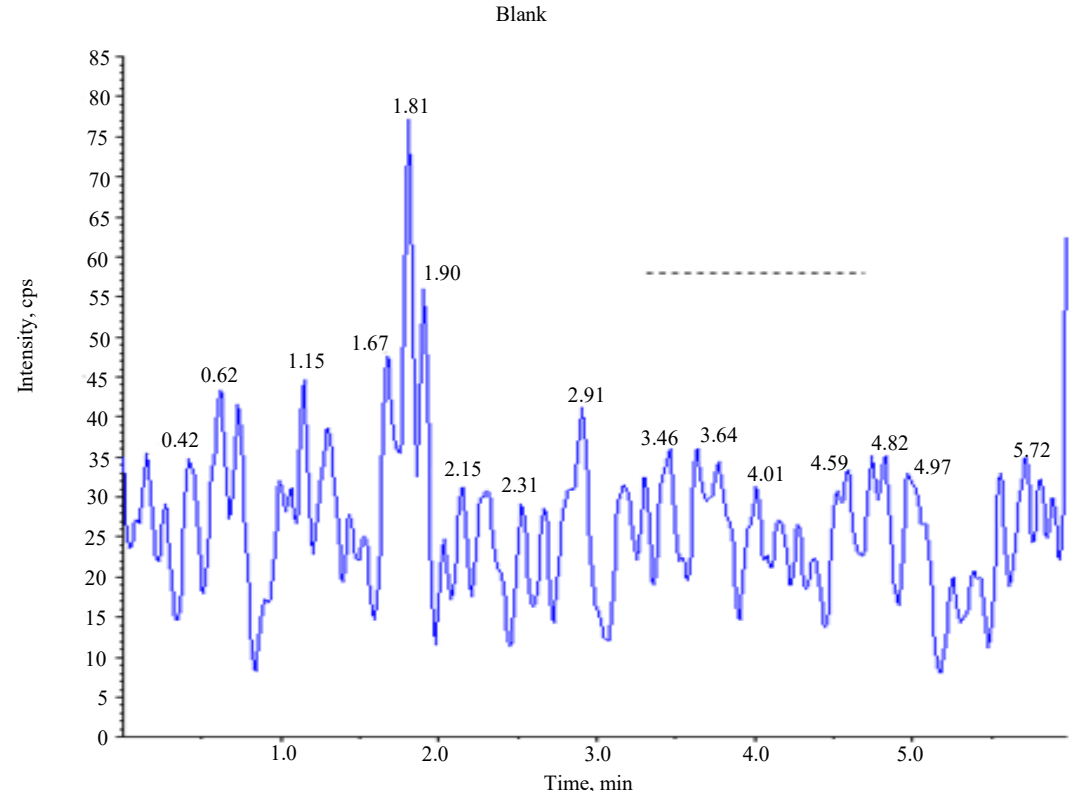



**Figure 2.** Peaks on blank and samples of patients.

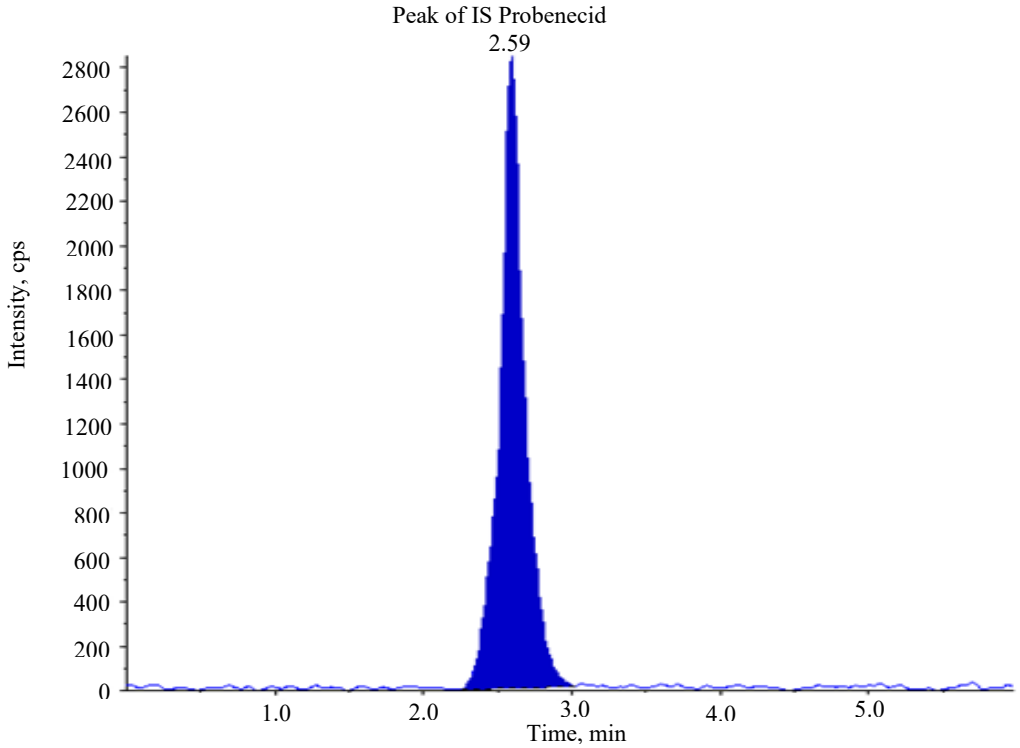
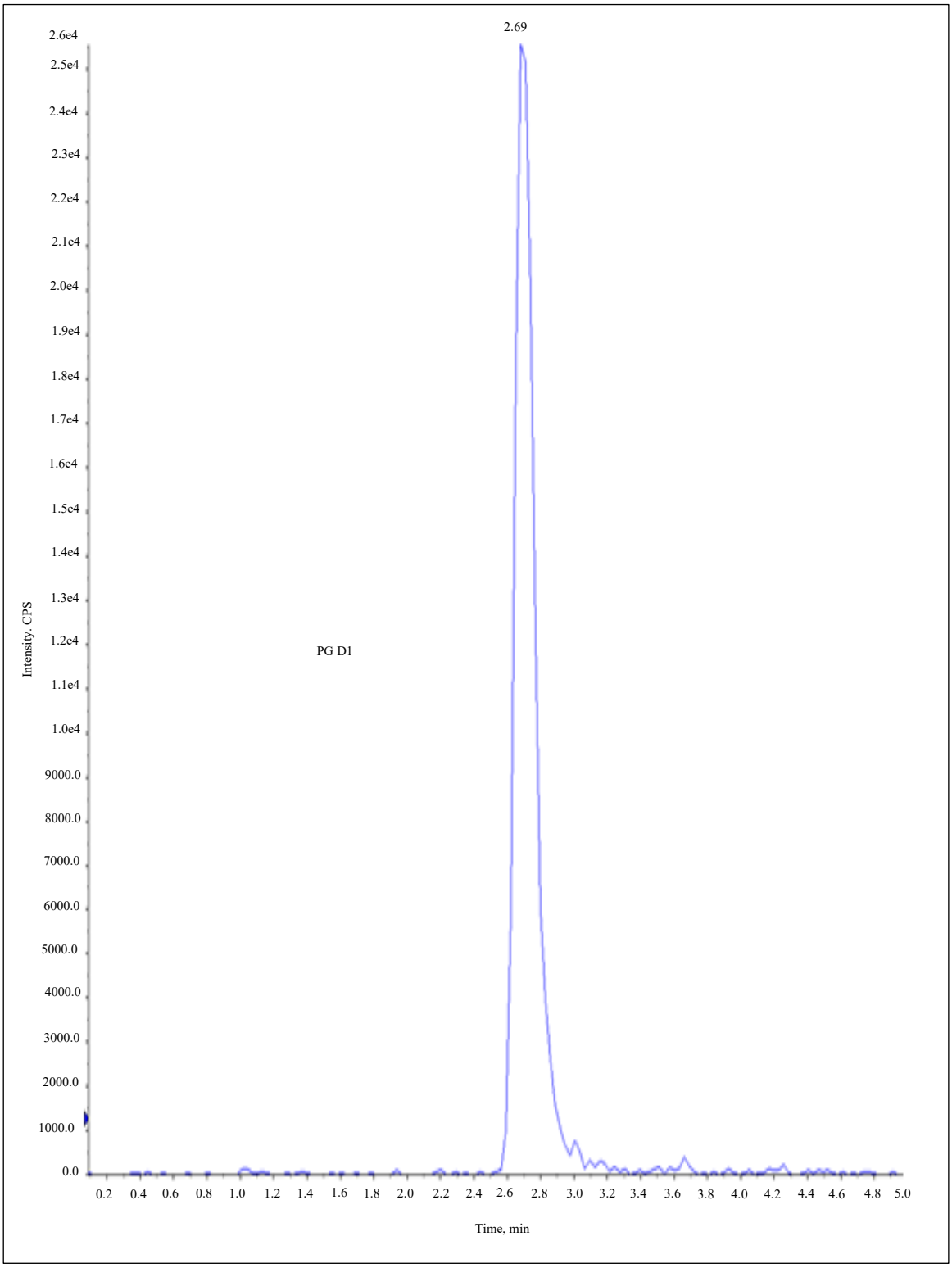



**Figure 3.** Standardization of methods the LC-MS/M peaks of standards made at 100 ng/mL.

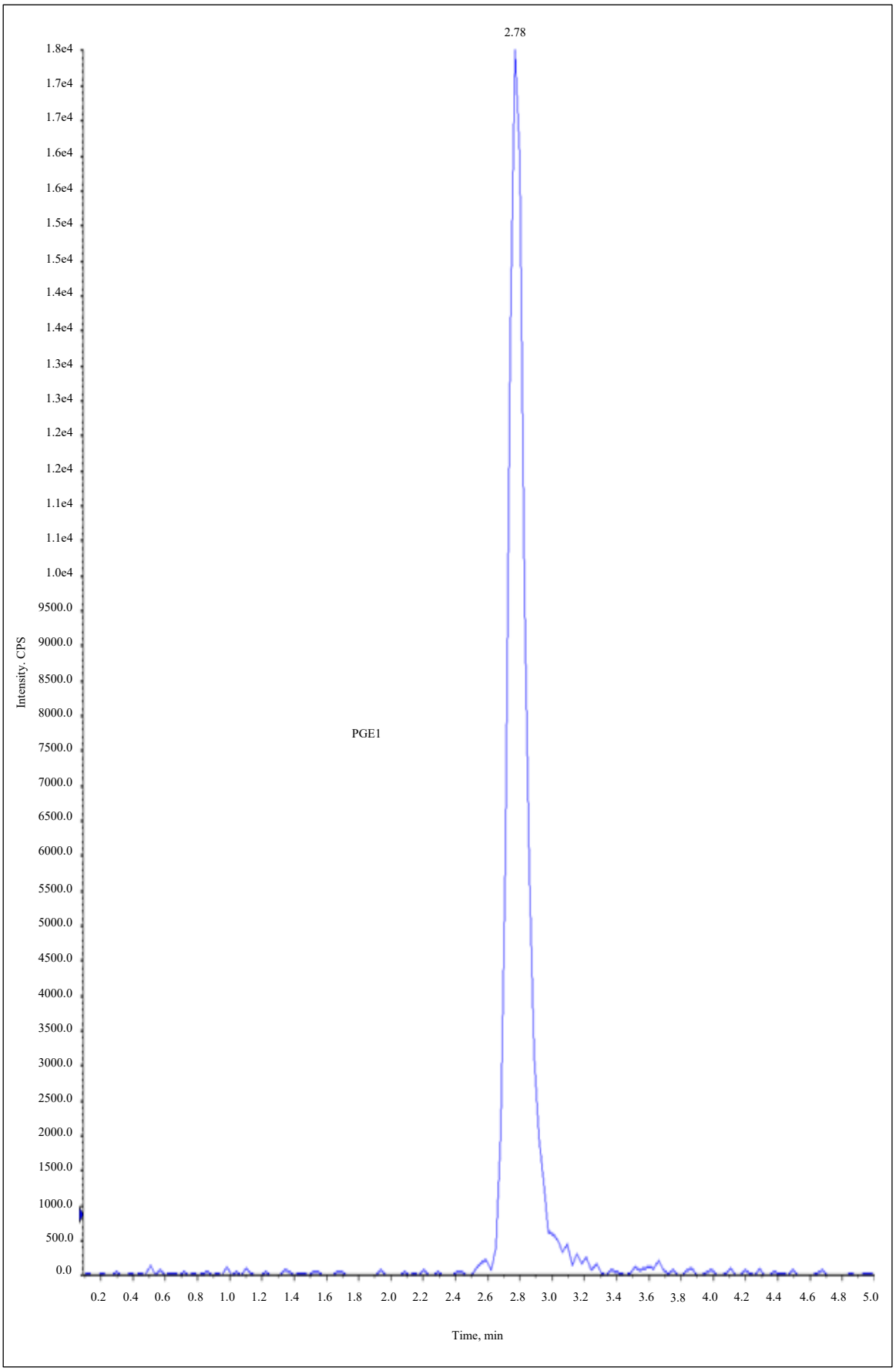
 

LC-MS/MS chromatograms of standard solution (100 ng/mL) showing distinct peaks of prostaglandins $PGD_2$, $PGE_2$, $PGA_2$, and $PGB_1$ along with the internal standard Probenecid, demonstrating method specificity and proper chromatographic separation (Figure 4) and in Figure 5, flow chart shows enrollment and follow-up status.

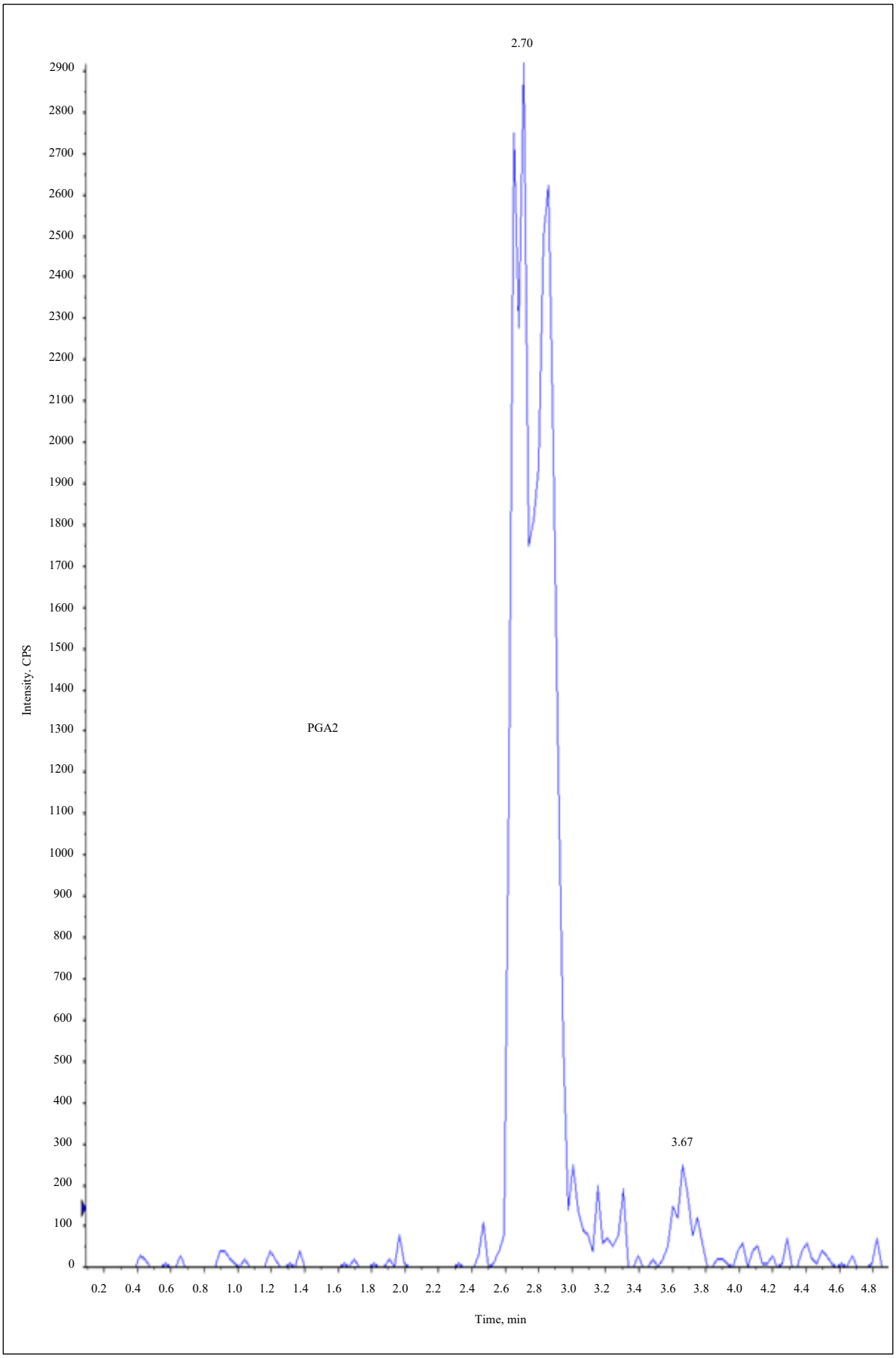
**LC-MS/MS PEAKS OF STANDARD 100NG/mL**



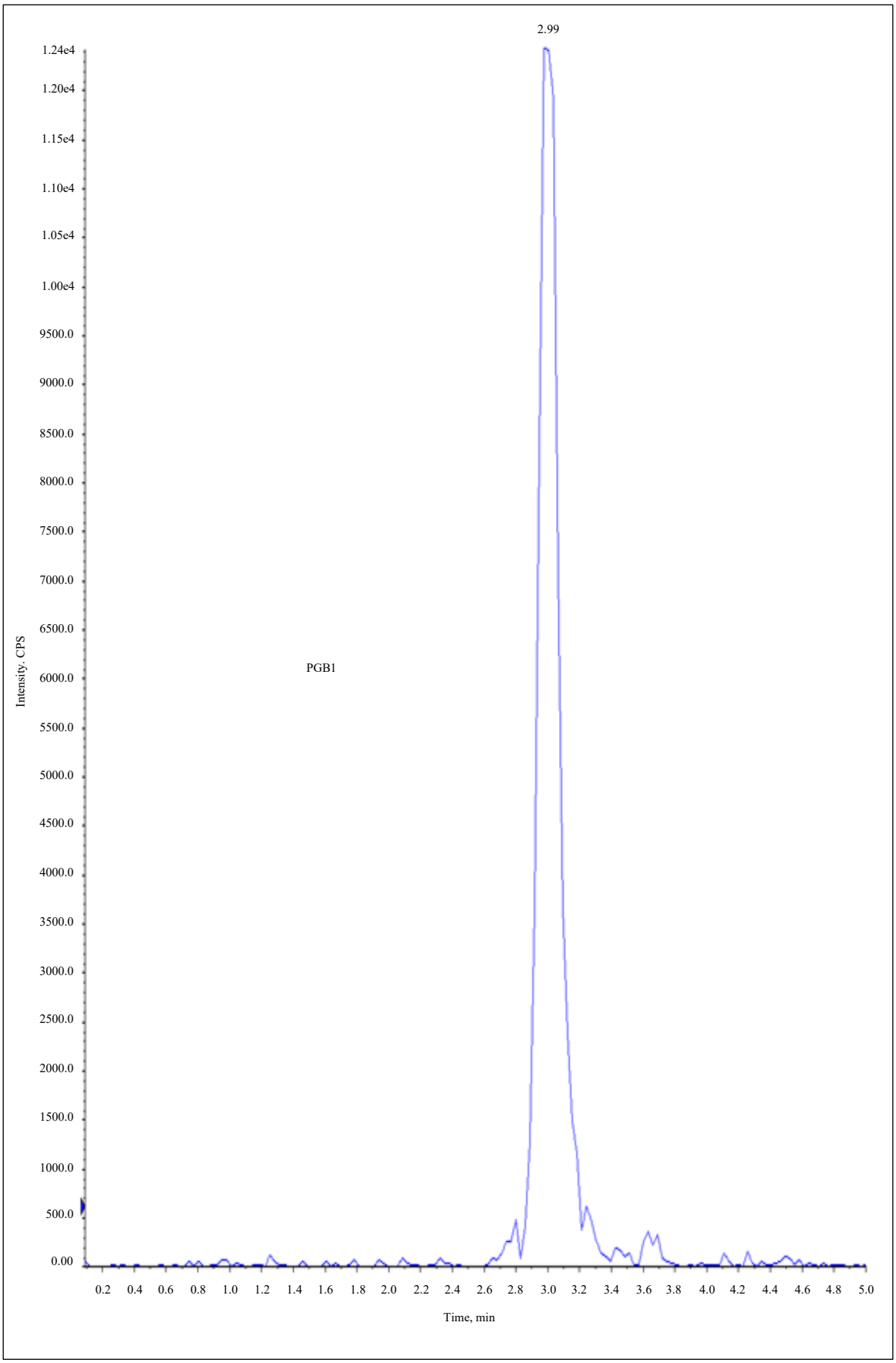

 







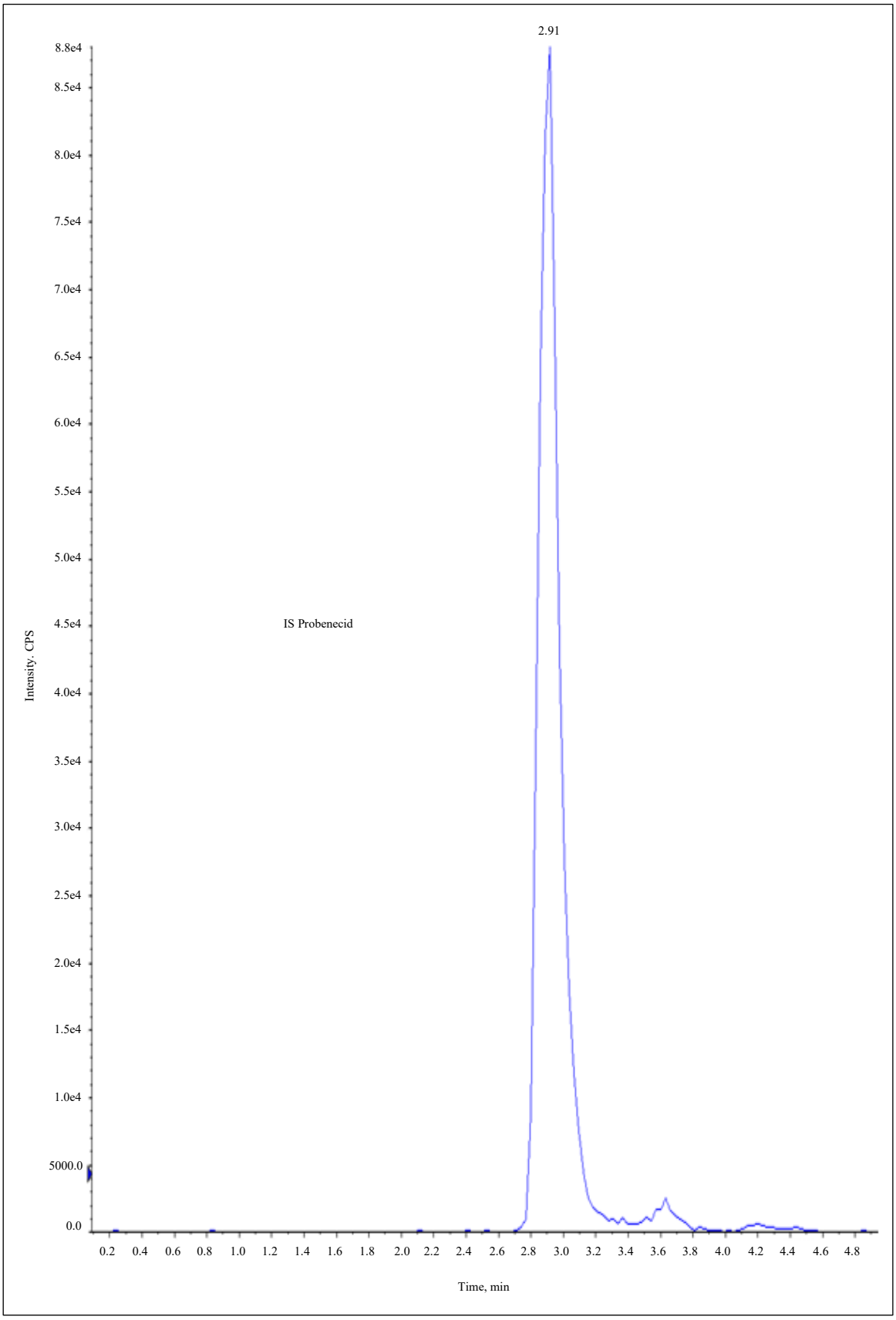



**Figure 4.** LC-MS/MS chromatograms of standard solution (100 ng/mL) showing peaks of $PGD_2$, $PGE_2$, $PGA_2$, $PGB_1$, and internal standard (IS) probenecid.

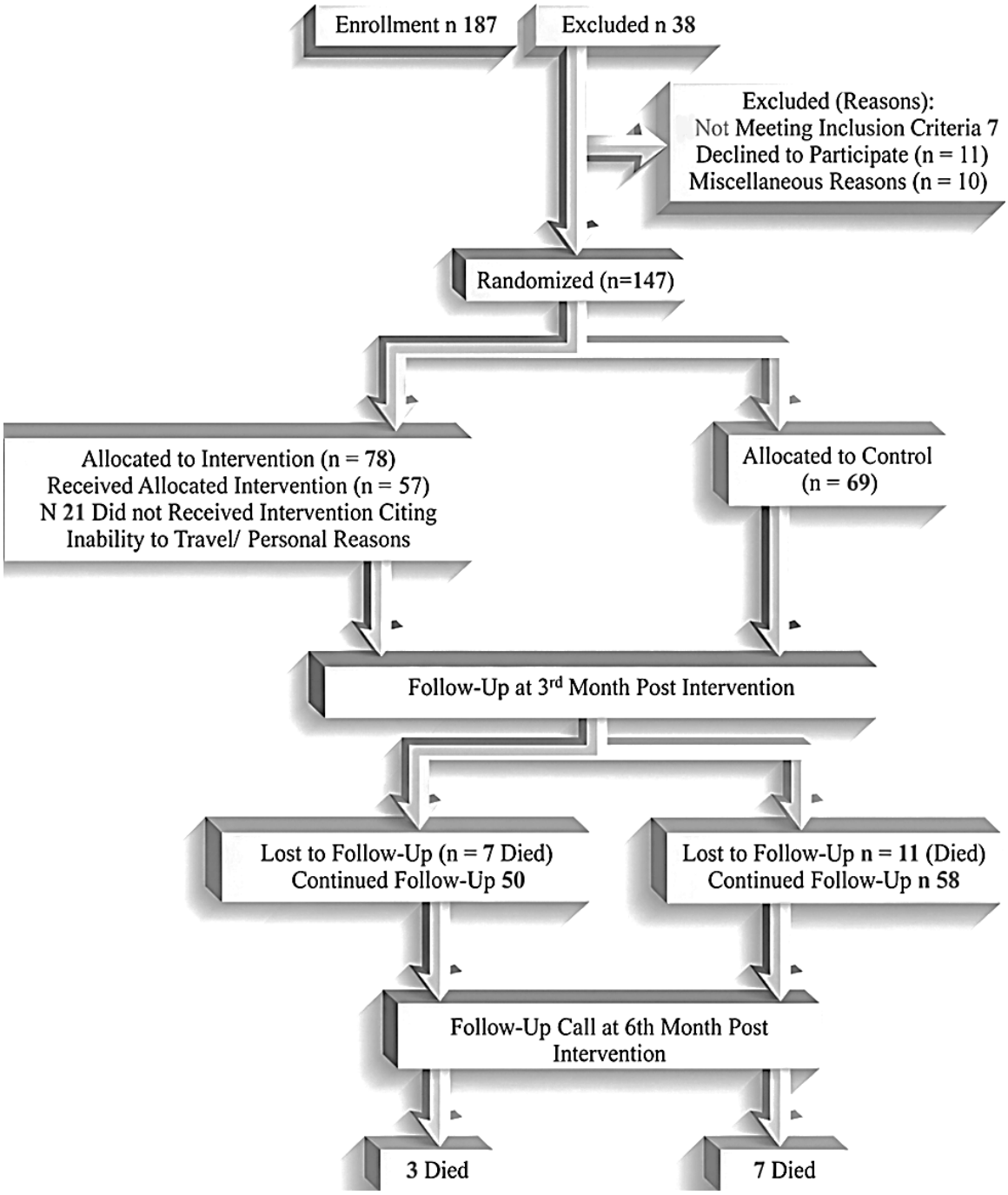



**Figure 5.** Flow chart above shows enrollment and follow-up status.

Intervention in standard care arm was management of pain and symptoms as per Institutes guidelines for palliative management. SKP arm plus standard care participants attended 4 days' workshop of 3 hours each day post-lunch. During course participants learned Sudarshan Kriya and life skills as per published standards and validated methods [30] under guidance of certified teachers in community center of hospital premises (Table 3).

Both the arms (standard care, standard + SKP) showed increased level of $PGA_2$ at 3rd month's sample which was more in SKP intervention arm, and the difference remained significantly high in SKP added arm (Table 4). $PGD_2$ & $PGE_1$ levels remained variable without significant difference – comparable in both the arms (Table 5).

**Table 3.** Demographic profile of participants study.

| Variables for demographic profile | SKP N 78 (%) | Control N 69 (%) |
|---|---|---|
| Age in completed years mean ± SD | 46.89 ± 9.49 | 48.22 ± 9.40 |
| Family history of breast cancer | 5 (6.4) | 3 (4.4) |
| ***Status of CA Breast at the Time of Enrollment*** | | |
| Clinically no evidence of disease | 3 (4.2) | 0 |
| Clinically active disease locally without metastasis | 29 (40.9) | 21 (29.4) |
| Clinically active disease locally with metastasis | 37 (52.1) | 46 (67.7) |
| Locally no disease but metastasis present | 2 (2.8) | 2 (2.9) |
| ***Education*** | | |
| Illiterate | 22 (29.3) | 22 (31.9) |
| Primary school certificate | 5 (6.7) | 12 (17.4) |
| Middle school certificate | 13 (17.3) | 13 (18.8) |
| High school certificate | 15 (20) | 11 (15.9) |
| Intermediate or post high school diploma | 4 (5.3) | 3 (4.4) |
| Graduate or postgraduate | 16 (21.2) | 6 (8.7) |
| Profession or honours | 3 | 2 (2.9) |
| ***Occupation*** | | |
| Unemployed/housewife | 68 (89.5) | 65 (94.2) |
| Unskilled worker | 3 (3.9) | 0 |
| Semi-skilled worker | 0 | 1 (1.5) |
| Skilled worker | 2 | 1 (1.5) |
| Clerical, shop owner, farmer | 2 (2.6) | 0 |
| Semi profession | 2 (2.6) | 1 (1.5) |
| Profession | 1 (1.3) | 1 (1.5) |
| ***Family Income Per Month*** | | |
| ≤1290 | 9 (12) | 11 (15.9) |
| 1291–6445 | 41 (25.3) | 29 (31.0) |
| 6446–9644 | 7 (9.3) | 8 (10.1) |
| 9645–12891 | 5 (6.7) | 5 (4.4) |
| 12892–25784 | 12 (16) | 13 (8.7) |
| 25785+ | 4 (5.3) | 3 (1.5) |
| ***Distance (km) Travelled Daily for Treatment*** | | |
| 0–50 | 57 (76) | 31 (44.9) |
| 51–100 | 2 (2.7) | 8 (11.6) |
| 101–600 | 11 (14.7) | 17 (24.6) |
| 601+ | 8 (6.7) | 13 (18.8) |
| ***Accompanying Person*** | | |
| Brother | 1 (1.3) | 4 (5.8) |
| Daughter | 4 (5.1) | 5 (7.3) |
| Husband | 44 (56.1) | 40 (58) |
| Sister | 2 (2.6) | 1 (1.5) |
| Sister-in-law | 0 | 1 (1.5) |
| Sisters' son | 0 | 1 (1.5) |
| Son | 12 (15.4) | 17 (24.6) |
| Public/neighbor | 1 (1.3) | 0 |
| None | 14 (17.9) | 0 |

**Table 4.** Levels of prostaglandins.

| Prostaglandin | SKP intervention+ regular treatment mean levels in nanogram/ml(n) | Control mean levels in nanogram/ml(n) | P value |
|---|---|---|---|
| ***Prostaglandin A2*** | | | |
| At baseline | 0.32 (38) | 0.22 (50) | 0.7 |
| At the 3rd month follow-up visit | 1.1 (30)↑ | 0.36 (38) | 0.0* |
| At the 6th month follow-up visit | 0.99 (25) | 0.56 (23) | .01* |
| ***Prostaglandin D2*** | | | |
| At baseline | 0.9 (38) | 0.7 (50) | 0.12 |
| At the 3rd month follow-up visit | 1.2 (30) ↑ | 0.8 (38) ↑ | 0.2 |
| At the 6th month follow-up visit | 1.2 (25) | 0.75 (23) | 0.5 |
| ***Prostaglandin E1*** | | | |
| At baseline | 0.22 (38) | 0.28 (49) | 0.38 |
| At the 3rd month follow-up visit | 0.3 (30) | 0.5 (38) | 0.13 |
| At the 6th month follow-up visit | 0.31 (25) | 0.34(23) | 0.4 |
| ***Prostaglandin E2*** | | | |
| At baseline | 0.9 (38) | 0.8 (50) | 0.07 |
| At the 3rd month follow-up visit | 0.83 (30)↓ | 0.32 (38) | 0.02* |
| At the 6th month follow-up visit | 3.3 (25) | 1.5 (23) | 0.3* |

*Note:* *– Statical Significance. ↑ – Value is Increasing ↓ – Value is Decreasing.

**Table 5.** Shows comparison of PGs level at 3 months post intervention.

| Prostaglandin | SKP + standard care vs. standard care | P value |
|---|---|---|
| PG A2 mean ± SD | 1.16 ± 0.19 vs. 0.36 ± 0.10 | 0.0 |
| PG D2 mean ± SD | 1.20 ± 0.73 vs. 0.87 ± 0.33 | 0.2 |
| PG B1 mean ± SD | 6.20 ± 3.2 vs. 10.2 ± 5.3 | 0.5 |
| PG E1 mean ± SD | 0.24 ± 0.6 vs. 0.28 ± 0.06 | 0.7 |
| PGE 2 mean ± SD | 0.8 ± 0.4 vs. 0.3 ± 0.6 | 0.0 |

There was significant rise in serum $PGA_2$ (1.16 ± SE0.19 vs. 0.36 ± 0.10, p value 0.0) among SKP intervention after 3 months of exposure to added intervention of SKP, it reduced at 6th months, still remained statistically significant high. This is the first ever report that shows one time intervention of SKP showing lasting effect till 3rd month post intervention while effect started declining by 6th months post intervention. It is important to mention that post intervention regular practice was not done by the participants as most of them were not staying nearby hospital and telephonic follow-ups gave idea that participants did not stick to regular practice schedules. Reason for noncompliance was told that either low motivation or forgetfulness prevailed once they returned home. Equivalence changes were observed for $PGB_1$ and $PGE_1$. The overall survival in intervention arm was 25 /38, 65.8% in SKP added arm and 23/50, 46.0% in control arm at 6th Months follow-up.

## DISCUSSION

Prostaglandins (PGs) are a family of biologically potent lipids from membrane phospholipids comprised of a $C_{20}$-unsaturated fatty acid containing a cyclopentane ring playing important role in cancer. Recent publications have shown that not all prostaglandins are pro-inflammatory and Prostaglandin $A_2$ ($PGA_2$) potently inhibits cell proliferation and suppresses tumor growth *in vivo*. Tumor suppressor Prostaglandin $A_2$ enhanced with the SKP added intervention in breast cancer showed for the first time the effect of any comprehensive breathing techniques impact on prostaglandin level. Level of prostaglandin in human blood from patients suffering from cancer has been explored recently to be used as a biochemical prognostic marker for determining their response to anti-cancer treatment [31]. Prostaglandins,

particularly prostaglandin $E_2$ ($PGE_2$), are potent inflammatory mediators derived from the cyclooxygenase (COX) enzyme pathway. A few studies on yoga have been shown to reduce general inflammatory markers like cortisol but none of them tested Prostaglandins. Studies done recently have found the pathway and mechanism of action of prostaglandins in cancer. More studies on SKP role in altering them will further enhance potential use of prostaglandins in cancer patient's follow-up.

This randomized controlled clinical trial was conducted during 2012–14 in the department of Onco-Anesthesia and Palliative Medicine under pain clinic in BRAIRCH, Institute of All India Institute of Medical Sciences, New Delhi. The patients of advanced stage breast cancer after completing chemotherapy, radiation, and surgery were attending the pain clinic for palliative management. The late reporting of this paper is mainly due to lack of staff and published explanations to observed findings. Recently published papers have explained role of prostaglandins in cancer prognosis [31]. The patients were tried to be contacted to update the status of survival; however, none of them found responding as most of them were from outstation, phone numbers were of relatives which were found changed. So, it is anticipated that after 10 years of post-intervention none of the participants is traceable.

Lacuna identified during the study was that patients enrolled were belonging originally too far of places quite away from the tertiary care center – outstation patients attending tertiary cancer care site – the study site. Due to distance patients did not practice regularly for what they were told in 4 days workshop. Hence home practice was mostly relied on the telephonic conversations, and when they came for 3rd and 6th month's follow-ups visits to give blood samples for routine tests as well as study biomarkers.

Practically, it is considered SKP as one time intervention with no regular practice by participants produced this much impact on their prostaglandin levels. Further studies with regular follow-up to ensure practice may give further insights. Despite only once exposure of SKP, the level of Prostaglandin $A_2$ ($PGA_2$) which potently inhibits cell proliferation and suppresses tumor growth *in vivo*/tumor suppressor were observed enhanced. Level of prostaglandin in human blood from patients suffering from cancer reflects to have potential for use as a biochemical marker for determining their response to anti-cancer treatment in this study as well in few more published papers.

## CONCLUSION

SKP as added intervention to terminal cancer cases enhances $PGA_2$ levels – potential cancer suppressor prostaglandins. Hence this intervention is recommended during cancer management.

**Acknowledgments**

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**Source of Support**

AYUSH, Ministry of Health.

**Conflict of Interest**

None declared.

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