THE HIGH COURT

2004 8001 P

BETWEEN

MARY HEALY

PLAINTIFF

AND

BRENDAN BUCKLEY

AND

THE BON SECOURS HOSPITAL, BON SECOURS HEALTH SYSTEM

DEFENDANTS

JUDGMENT of O'NEILL J. delivered on the 20th day of May, 2010

1. Factual Background

1.1 The plaintiff, in this case, sues the defendants for damages for negligence and breach of duty in the treatment of the plaintiff between September 2000, and March 2001, with the drug known as Sandostatin LAR.

1.2 The first named defendant is a consultant Endocrinologist at the second named defendant's hospital.

1.3 The plaintiff was born in 1946, and is a married woman with three adult children.

1.4 In February 1982, the plaintiff was found to have a large pituitary tumour. Initially, she declined surgery for this. Later on in the year in September 1982, she was admitted to hospital, quite distressed, with severe pain, headache and photophobia. A neurological examination revealed complete left-sided opthalmoplegia with absence left corneal reflex. Fundoscopy showed early papilloedema, with early evidence of optic atrophy.

1.5 The plaintiff was pregnant at the time. A CT scan showed an enlarged pituitary intrasellar tumour extending to the left side with evidence of haemorrhage in this area. A carotid angiogram showed a large central mass effect with distortion of the internal carotid and middle cerebral arteries. Initially, the plaintiff was given drug therapy. Her condition deteriorated and it was decided to remove as much of the tumour as possible.

1.6 On 29th September, 1982, a left frontal temporal craniotomy with biopsy of the pituitary tumour was performed. Because of the vascularity of the tumour, only a very limited biopsy was taken. This showed a small amount of infarcted tissue. Following surgery, she developed a left ptosis.

1.7 The plaintiff improved following her surgery and was continued on the medication she was on before the surgery i.e. Bromocryptine, and she was also prescribed Carbamazepine for temporal lobe seizures which had developed as a result of the pressure of the tumour on the left temporal lobe.

1.8 In 1984, the plaintiff's treating surgeon, Mr. Feely, thought the tumour was slightly larger in spite of the Bromocryptine. The plaintiff continued on Bromocryptine and has done so ever since. The plaintiff continued under the care of Mr. Feely until he left his post in 1992, and thereafter, she came under the care of Mr. Charles Marks, a neurosurgeon, and also a little later, Dr. Teresa Mitchell, a consultant Endocrinologist.

1.9 By the early 1990s, it was clear that the plaintiff had an extremely rare tumour which was hyper-secreting three hormones, namely, Thyroid Stimulating Hormone (TSH), Growth Hormone (TH) and Prolactin. This is not only an extremely rare condition, but in the experience of all of the experts who have given evidence in this case, unique.

1.10 From an early stage, the excessive secretion of Prolactin was successfully suppressed by the Bromocriptine. Much greater difficulty was encountered in dealing with the excess secretion of TSH and GH. Dr. Mitchell treated the excess secretion of TSH and the consequent Thyrotoxicos with Propranol and Carbimazole and her seizures were treated with Carbamazepine.

1.11 In 1994, various treatments were considered for the plaintiff. Primary of these was radiotherapy, but it takes many years, frequently up to ten or more years for the beneficial effects of this treatment to take hold. The drug type Somatostatin or its analogue, Octreotide, was considered. Sandostatin, the drug at issue in this case, is that type of drug.

1.12 In 1994, it was available, but in a different format to the one at issue in this case. Then, the drug had to be administered by three daily injections into the abdomen. The treatment given to the plaintiff in these proceedings, in September 2000, was a long-acting version of the drug which became available in the late 1990s, and is given by means of a single injection every four weeks into the gluteal muscle.

1.13 The Cork University Hospital notes indicate that around February 1994, the Somatostatin type drug was considered but the plaintiff was not keen on it. Also, at the time considered was radiotherapy and the plaintiff opted for this treatment which was carried out that year. In the meantime, the plaintiff continued on quite high levels of Bromocriptine and anticonvulsant medication.

1.14 Pending the effects of the radiotherapy on the plaintiff's Growth Hormone levels and her Thyroxine, Dr. Mitchell increased her Bromocriptine to 10mg. per dose. In the spring of 1994, the plaintiff had a radical course of radiation of her tumour over a period of six weeks. As of January, 1995, Dr. Mitchell found that a CAT scan demonstrated no further growth of the tumour and that her Growth

Hormone levels had stabilised but her serum Thyroxine, though much lower, was still abnormally high. She also found that her serum cortisol had fallen either due to the tumour or to the radiotherapy.

1.15 During 1995, the plaintiff decided to leave the care of Dr. Mitchell and her

GP referred the plaintiff to the first named defendant who saw her first in

September 1995. The first named defendant admitted the plaintiff to the

second named defendant's hospital for a full workup to establish her pituitary

status. In addition, he referred her to the Radiological Department of St.

Vincent's Hospital in Dublin for a MRI scan of the tumour. The report of this

MRI scan stated its findings as follows:

> *"A large multi-lobulated enhancing mass lesion is present within the pituitary fossa with extensive spread outside of the pituitary fossa. This lesion measures 4cms x 3 cms x 3cms. The lesion has grown through the floor of the pituitary fossa into the left side of the sphenoid sinus and demonstrates extensive left-sided parasellar and suprasella extension. The lesion is immediately adjacent to the medial aspect of the left temporal lobe which it has compressed. Anteriorally, the lesion has encased the left internal carotid artery. There is no apparent compression of the optic chiasm or hypothalamus. The pituitary stalk is displaced towards the right side. There is no evidence of hydrocephalus."*

1.16 As a result of biochemical review, the first named defendant put the plaintiff on a cortisol replacement and continued her on other medications. The first named defendant reviewed the plaintiff in October 1996, and found her to be doing well. Her biochemistry, as of this time, was satisfactory, so no alteration was made in her medication.

1.17 The plaintiff was next reviewed by the first named defendant on 22nd July, 1997. At that time, she was symptomatically well. The plaintiff was troubled by her left eye ptosis and thereafter, the first named defendant referred her to Dr. Ger O'Connor, an ophthalmic surgeon. Her biochemical tests were satisfactory. She was seen again by the first named defendant on 16th February, 1998. She appeared to have been well then, although reported having 'flu over Christmas. She complained of early morning wakening. Her hormonal situation was as before.

1.18 In November 1998, the plaintiff had an Endocrine review. A chest X-ray of 10th November, 1998, showed minor cardio megaly. Her routine biochemistry and haematology done then were unremarkable. She was, however, excreting excess TSH, causing her to be slightly hyperthyroid. The plaintiff was next reviewed by the first named defendant on 2nd March, 1999, when there appears to be no change in her condition.

1.19 In April 1999, she had surgery on her left eye to deal with the ptosis. The outcome of this surgery appears to have been good.

1.20 The plaintiff was next reviewed by the first named defendant on 29th November, 1999. Nothing remarkable appears to have transpired from this review.

1.21 The plaintiff next encountered the first named defendant on 24th July, 2000. The first named defendant was of the view, that as it had been some time since the plaintiff had had a full review, he proposed bringing her into hospital for a couple of days to do this, including getting an MRI scan done.

1.22 It would appear that a consultation took place between the plaintiff and the first named defendant on 24th July, 2000, at which or after which, the first named defendant filled out a booking form for the second named defendant's hospital with an admission date of 14th August, 2000. The plaintiff was admitted to the second named defendant's hospital on that date and had a series of tests carried out to assess the status of her pituitary tumour.

1.23 On 15th August, 2000, she had an MRI scan carried out in Cork University Hospital. The report of the radiologist on this MRI, in its findings and conclusion, reads as follows:

> *"Findings: previous left craniotomy. There is a very large enhancing pituitary mass measuring about 4cms in maximum diameter, displacing the pituitary stalk to the right. The mass involves the residual part of the pituitary gland and extends into and lateral to the left cavernous sinus, completely surrounding the internal carotid artery. The mass extends inferiorly into the sphenoid sinus and laterally compresses the left temporal lobe, its most posterior portion extends along the left petrous apex.*
>
> *Conclusion: very large enhancing pituitary mass involving the left cavernous sinus. Appearances are consistence with recurrence of the patient's pituitary tumour."*

1.24 The radiologist who carried out the MRI, Dr. A. Brady had not been furnished with the radiologist's report from the previous MRI in 1995 and hence, could not compare one with the other. It is accepted by all of the expert witnesses in the case and also the first defendant that the August 2000 that the MRI did not show any significant change to the 1995 MRI. As Dr Brady did not have sight of the earlier MRI to compare he naturally concluded that there had been a reoccurrence of the plaintiff's pituitary tumour.

1.25 Amongst the tests carried out on the plaintiff in hospital on this admission was what is known as a IGF-1 test and also a Glucose Tolerance Test (GTT). This latter test involves the testing of growth hormone levels by way of an interaction with glucose administered at fixed intervals over the course of a fixed period of time.

1.26 In the case of the plaintiff, three testings were provided for within the overall scheme of this test. Unfortunately, due to an error, this latter test was not fully reported on and the only outcome of it was one growth hormone result which eventually was reported on 24th October, 2000 and showed a growth hormone level of 6.7. The purpose of both of these tests is to show growth hormone levels.

1.27 The IGF-1 test result came back to the first defendant on 5th September, 2000 and this showed the growth hormone level significantly above the normal range. It was reported at 514, whereas the normal range is 107 – 310. Thus, the result was

approximately 70% above the highest point in the normal range. Much controversy occurred in the evidence as to the significance of these test results. The first named defendant was very concerned at the elevated IGF-1 test result and it was very much part of the subject matter of a telephone conversation he had with the plaintiff on the same day. The test in question was considered by the first named defendant as a reliable indicator of hyper secretion of growth hormone which greatly concerned the first defendant because of the serious health consequences, of a persistently elevated growth hormone level. When the first defendant telephoned the plaintiff the growth hormone test result from the GTT had not been returned with the result that the first named defendant based his conclusions and recommendations solely on the IGF-1 result. For this he was heavily criticised by the expert witnesses called by the plaintiff. I will deal with that aspect of the case later. The IGF-1 test result is a more consistent measure of growth hormone levels which tend to fluctuate during the course of the day. Growth hormone is released in pulses usually during the night and it is also released in response to a variety of stresses thus depending upon when a single growth hormone test is taken, very different results can be obtained. The Glucose Tolerance Test attempts to even out these fluctuations by doing the test at a number of different times during the day. The IGF-1 test, however, is a measure of a different substance produced in response to growth hormone and has a much more consistent presence.

1.28 In the meantime, on 5th September, 2005, the IGF1 test result was reported. When this test result was received, the second named defendant rang the plaintiff to discuss the test results with her. Over the three-week period since she had been in hospital, the plaintiff had phoned on a number of occasions enquiring of her test results. In the course of this telephone conversation, the second named defendant disclosed to the plaintiff the results of her tests. Essentially, these were twofold, namely, that the MRI scan revealed that she still had a large pituitary tumour, and secondly, that her growth hormone level, as revealed by the IGF1 test was very significantly elevated.

1.29 There is no doubt that the outcome of this telephone conversation, from the point of view of the professional relationship between the plaintiff and the first named defendant, was most unfortunate. A very large part of the difficulties that arose stemmed from the fact that the plaintiff, prior to this telephone conversation, believed that her tumour had been, in effect, removed, leaving only something the size of the tip of her finger, and it came as a great shock to her to be told that she had a tumour of the size as disclosed in the MRI scan, and it is clear that what she took from the information that was given to her by the first named defendant was that the tumour had started to grow again and was progressing rapidly.

1.30 I am quite satisfied, from the evidence, that the first named defendant did not tell the plaintiff that the tumour was progressing or growing, but that he did tell her that the tumour was very unsatisfactory.

1.31 There is no doubt he also told her about the excess secretion of growth hormone, as revealed in the IGF1 test, and I am quite satisfied that information, combined with the information about the size of the tumour, greatly alarmed the plaintiff and conveyed to her a picture of a very serious and dangerous deterioration in her health.

1.32 The second named defendant, on the other hand, sought to present to her this state of affairs in her treatment in a light which was very positive, in the sense that he conveyed to her that the elevated growth hormone level presented an opportunity to use the drug, Sandostatin LAR, which had relatively recently become available, and which was effective in reducing excess growth hormone levels and it was to be hoped, also would reduce the size of the tumour.

1.33 From the first named defendant's point of view, it was the high IGF1 test result which provided the indication for the appropriate use of this drug. The first named defendant saw this test result as triggering an opportunity, because of the availability of this drug to intervene for the first time since the plaintiff had radiotherapy so as to render the tumour safer to live with for the plaintiff.

1.34 Because of the plaintiff's distaste for injections and her rejection at earlier time of the Sandostatin s.c., no other treatment had been available which could appropriately be deployed which might have had a beneficial effect on the plaintiff's tumour, until that time.

1.35 In the course of the conversation, the second named defendant mentioned his experience with the drug, Sandostatin, and of its successful use, and whilst in no way guaranteeing a successful outcome, I am satisfied he did vouch for the drug in the sense of very positively recommending it for the plaintiff.

1.36 The first named defendant cannot be blamed or faulted for the state of mind of the plaintiff concerning her tumour at that time. No doubt, as he said in evidence, having regard to the length of time the plaintiff had this condition, and the amount of tests and scans that had been done on it and the amount of consultation she had with him and with other doctors, he thought that she was well informed as to the state of her tumour.

1.37 The first named defendant sent a prescription for this drug to the plaintiff by post. I am quite satisfied that the plaintiff did not send it direct to a chemist shop, as contended by the plaintiff. He was nor aware of who was the plaintiff's chemist and the box on the prescription form for the name of the chemist is not filled in. The first named defendant also communicated with the plaintiff's GP who was to administer the injections and subsequently wrote to the plaintiff's GP by a letter of 14th September, 2000. This letter was very much in controversy in the evidence and reads as follows:

> *"Dear Len,*
>
> *I admitted Mrs. Healy for routine follow up.*
>
> *The most important features are that her MRI scan shows that her pituitary tumour is still very unsatisfactory in size and shape as it extends beyond the sella and is quite close to her optic nerve.*
>
> *On this occasion, her IGF1 is back elevated to about three times the upper end of normal, and accordingly, I think that she should start on Octreotide LAR 'the High Tech' scheme. I have sent her a prescription for this. She will need this to be injected into her Gluteus every four weeks. If you find this is a problem, please let me know, and we can see what we can organise otherwise.*
>
> *Incidentally, her other routine endocrine tests were satisfactory with free T420, TSH2, Prolactin 9.8, FSH 2.2, LH 1.2.*
>
> *Her DEXA scan shows a Z score of -0.9 (T score -2.6) indicating mild osteoporosis on the lumbar spine, but the density in her femoral neck was excellent with an above average Z score of 0.7 and a T score of -0.5.*
>
> *I am naturally relieved to see that her bone density is so good in view of the potential for problem posed by her*

endocrine problems up to now, and no action is required at this stage but we should repeat the DEXA scan in about three years.

My experience with growth hormone secreting tumours, such as this, is that they are now very responsive to this new agent. I would plan to review her with a new MRI in six to twelve months."

1.38 It was contended, on behalf of the plaintiff, that the letter demonstrated that the first named defendant was of the view that the plaintiff's tumour had increased in size since the previous MRI was done in 1995. This contention was supported by reference to the conclusion in the radiologist's report of the MRI done on 15th August, 2000.

1.39 I am quite satisfied that the first named defendant was not at all under the impression that the MRI done in August 2000 disclosed a progressing tumour. The use of the word "*still*" in his letter of 14th September, 2000, indicates that he was mindful of the state of the tumour disclosed in the 1995 MRI, but regarded the continuation of that state as disclosed in the August 2000 MRI, as unsatisfactory.

1.40 Much criticism was levelled at the first named defendant because, in this letter, he described the result of the IGF1 test as disclosing the growth hormone level at being three times the normal level. It is, of course, apparent that the result of the test at 514 was manifestly not three times the normal level, but approximately 70% of the highest point in the normal range.

1.41 I accept the first named defendant's explanation of this as an inadvertent error and that there was no conscious or deliberate attempt to mislead.

1.42 Soon after the administration of the first injection on 8th September, 2000, the plaintiff began to experience distressing vomiting and diarrhoea and she got in touch with the first named defendant by telephone and a consultation was arranged.

1.43 The first named defendant saw the plaintiff in his office on 18th September, 2000, and the outcome of that consultation was that the first named defendant altered the dosage of the drug to reduce it by half, from 20mgs to 10mgs, and he wrote out a prescription to that effect on that occasion, addressed to the chemist nominated by the plaintiff.

1.44 The plaintiff had the second injection at the lower dose at the beginning of October 2000. Shortly thereafter, the plaintiff was admitted to the Bon Secours Hospital for a surgical dental extraction. This was done under general anaesthetic. The hospital notes for the plaintiff's stay do not indicate any complaint by the plaintiff of gastrointestinal problems during her time in hospital.

1.45 The plaintiff had the third injection, as planned, approximately four weeks later. At the end of November, i.e. on 30th November, 2000, the plaintiff was again admitted to the Bon Secours Hospital to have a D&C procedure carried out. Her admission appears to have coincided very closely with the fourth injection of Sandostatin LAR. Whilst in hospital for this procedure, the hospital notes do not disclose any complaint by the plaintiff of gastrointestinal problems and, specifically, they would appear to have been a negative response to a query concerning bowel problems. No request was made of the first named defendant to attend the plaintiff during either of the two hospital admissions in October and November 2000.

1.46 It is quite clear that by the end of December 2000, the plaintiff had become quite ill, such that she was referred to hospital by her GP, Dr. Harty, and was admitted to the Bon Secours Hospital on 2nd January, 2001, complaining of severe vomiting and diarrhoea for some time, severe exhaustion, headaches, cramps and muscular spasms in her arms and legs.

1.47 The plaintiff's encounter with the first named defendant whilst in hospital on this occasion would appear to have been very unsatisfactory from the plaintiff's point of view, and also, the first named defendants.

1.48 The plaintiff reacted very badly to a suggestion by the first named defendant that she might have been suffering from depression and her evidence was that he met her in the corridor and handed her a prescription for an antidepressant. The first named defendant categorically denies in evidence dealing with the plaintiff in this manner.

1.49 Following her initial encounter with the first named defendant, the plaintiff became very angry and frustrated and decided to discharge herself from the hospital, but, obviously, did not proceed with this.

1.50 An Ultra Sound was ordered by the first named defendant and this was done the following day to test for gallstones, but the results of this test were negative.

1.51 On 4th January, 2001, prior to her discharge at the first named defendant's request, and with the plaintiff's permission, the first named defendant had a lengthy consultation with the plaintiff's husband and the plaintiff's sister at which the advisability of continuing with the Sandostatin LAR injections was discussed. The Plaintiff had not taken the injection due at the beginning of January 2001 i.e. the fifth injection.

1.52 While the plaintiff had been in hospital, routine blood tests were done, and these revealed that the plaintiff had become severely hypothyroid.

1.53 Following the plaintiff's discharge, the first named defendant, by letter of 5th January, 2001, wrote to the plaintiff's General Practitioner in the following terms:

"Dear Len,

Thanks for referring Mrs. Healy back with diarrhoea from the sandostatin. She also complained of muscle cramps, headaches, exhaustion and a sensation of pounding pulse, occasionally near her left ear when in bed. She had a marked sleep disturbance and had episodes of weepiness. As you know, she has a very large pituitary tumour extending beyond the sella adjacent to her optic nerve.

On examination: she had put on further weight. I felt, on mental state examination, that she was depressed, and this is not surprising in view of the considerable number of life events in the recent past. There was nothing else of note.

Investigations: Ultra Sound and gall bladder showed no evidence of gallstones. Routine biochemistry and haematology were unremarkable. However, interestingly, her thyroid function increased substantially with a Free T3 less than 1.7. Her TSH, of course, is only 0.51, and this represents hypothyroidism secondary to failure of her pituitary thyrotroph.

Interestingly, on her last review, her Free T4 being 20. Prolactin High N was completely suppressed and on measurably low level, probably as a result of the sandostatin which is unlikely to have caused her to become hypothyroid.

She expressed some considerable dissatisfaction of the diagnosis of depression and was adamant she would not take an antidepressant. She may come around to this in due course. With her permission, I discussed her case with her husband and sister. She will consider whether she wishes to continue in my care.

Plan: I have suggested that we should refrain from using sandostatin for another four weeks, but consider its recommencement around 1st February, or so. In addition, I have posted her a prescription for Thyroxine 50 micrograms for one week, increasing to 100 micrograms for a second week and then moving to a continuous maintenance of 150 micrograms daily. I will review her in about six week's time, if she wishes.

. . ."

By letter of the same date, the first named defendant wrote to the plaintiff as follows:

"Dear Mrs. Healy,

On the day after your discharge, results of your thyroid hormone levels were reported to me. These show that you now do not produce enough thyroid hormone. This is because your pituitary has stopped signalling the thyroid gland to make appropriate amounts of the thyroid hormones. This will certainly have contributed to some of the symptoms which made you feel unwell over the last few weeks. The failure in your thyroid hormones has occurred very recently, as up to now, the levels were always normal.

A similar thing occurred in the past with your production of cortisone which meant that we have to supplement you by giving you Hydrocortisone tablets. Your thyroid hormone results show that we need also to replace thyroid hormone and this is in the form of a tablet called L-Thyroxine and I enclose a prescription for this. This should be taken in addition to your medicines and is completely compatible with all of them. You take one tablet per day for a week. On the second and third weeks, take two tablets a day. From the fourth week onwards, take three tablets a day, regularly. I would like to see you in the Cork clinic on Tuesday 20th February, 2001, at 4.45pm, if you wish.

I suggested to Dr. Harty that you might recommence the sandostatin in around the first week of February.

Your thyroid hormone failure is an anticipated long-term effect of pituitary radiotherapy. It always occurs sooner or later in all patients who have had radiotherapy pituitary tumours, usually coming on between five to ten years after the treatment, as it has in your case. It is definitely not a side effect of sandostatin. . . ."

1.54 The first named defendant, in his evidence, now accepts that the plaintiff's hypothyrodism was in fact a side effect of the Sandostatin LAR but was not, at the time the plaintiff underwent this treatment, a known side effect of the drug.

1.55 As the plaintiff's GP was unwilling to administer any further injections of Sandostatin LAR to the plaintiff, the plaintiff sourced a nurse who was known to her who gave her the last two injections, the first of these at the beginning of February, and the final one at the beginning of March 2001.

1.56 The plaintiff attended the second named defendant at the Cork Clinic on 20th February, 2001, with her husband. She complained of having a severe bout of vomiting and diarrhoea and abdominal pain, two weeks after the Sandostatin injection at the beginning of February.

1.57 On 27th February, 2001, the first named defendant wrote to the plaintiff's GP, Dr. Harty, in the following terms:

"Dear Len,

I reviewed Mrs. Healy. She had single bout of diarrhoea about a fortnight after her last sandostatin injection, but this was very short lived, and I am pretty sure it was caused by the endemic infective agent that is going around at the moment, causing this widely. We discussed the options for treatment. I reassured her that her Hydrocortisone and Thyroxine replacement was absolutely necessary because of her deficiency in production of these hormones herself. She will not contemplate radiotherapy and surgery will not remove what is now a very complex pituitary tumour. I therefore pointed out that sando-statin was the only remaining option.

I saw her with her husband and they both said that they would go away and consider what we had spoken about. I would advise that she continues on sandostatin monthly, in the long-term, and I would plan to see her in three months time, at which stage I would take blood for IGF-1, Prolactin and T4. I pointed out that there was no point in repeating the MRI in under a year, particularly as it is not going to alter our management . . ."

1.58 An appointment was made for the plaintiff to come back to the first named defendant on 8th May, 2001, but the plaintiff did not attend for this appointment, having decided to discontinue her care by the first named defendant. She was, thereafter, referred by her GP to another endocrinologist, Dr. Howel Walsh.

1.59 In April 2001, Dr. Walsh admitted the plaintiff to Shanakeil Hospital for tests. By letter of 13th May, 2001, to the plaintiff's GP, Dr. Walsh discussed the results of those tests in the following terms:

"Dear Len,

I took Mrs. Healy back into Shanakeil again, and the results of our investigations are as follows:

IGF1 (done in April) 274

UG/1, which is within the normal range, this was taken a month after a previous dose of Octreotide

Free T4 (while off Thyroxine) 7.5 PMOL/1

Free T3 normal

TSH low at 0.25 MIU/1 (as one would expect in secondary Hypothyroidism)

I await Growth Hormone profile, repeat IGF1 and some other results.

Thus, it does seem as if this lady does have secondary Hypothyrodism, though her normal Free T3 is a little surprising. Under the circumstances, I recommend Thyroxine in a dose of 50 ugs. daily, and further increases may be necessary.

I await Growth Hormone profile to determine whether or not she is secreting excess Growth Hormone, as was thought to be the case in the past. She is also concerned about some retrosternal choking and this has lessened to some extent since discontinuing the Octreotide.

I have suggested the following:

(a) We are discharging her on Thyroxing 50 ugs. daily, and of course, she continues on Natrilix, Estrapak, Hydrocortisone 50 mgs in the morning and 5 mgs . . .”

A further letter was written by Dr. Walsh on 30th May, 2001, in which he says:

“Dear Len,

Further results for this lady are as follows:

Growth Hormone profile shows a fasting Growth Hormone of 1.5 MIU/1 and a midday value of less than 0.5 MIU/1.

IGF 1 was well within the normal range at 265 UG/1 (normal 107-310).

The present findings certainly do not suggest that there is a hyper-secretion of Growth Hormone and, for the moment, I think it is reasonable to defer reintroducing Octreotide. . . ”

1.60 Later in 2001, the plaintiff had a further MRI scan of her pituitary tumour carried out. This showed a ten per cent shrinkage in the tumour, as compared to the MRI done in August 2000.

2. The Issues

2.1 The first matter that must be confronted is whether or not the recommendation by the first named defendant to the plaintiff to take the Sandostatin LAR drug, in September 2000 and thereafter, was a breach of the first named defendant’s duty of care to the plaintiff.

2.2 The second issue is whether or not the plaintiff’s consent to accept the foregoing treatment was a valid informed consent or was it vitiated by a lack of sufficient information and/or misrepresentation or misinformation concerning the plaintiff’s condition at that time, and the appropriateness of Sandostatin LAR as a treatment for it.

3. **Was Sandostatin LAR an appropriate and acceptable treatment for the plaintiff’s condition in September 2000?**

3.1 Before commencing on the analysis of what was done by the first named defendant, it is worthwhile to bear in mind the nature of the duty and the standard of care that was required of the first named defendant. The head note to the case of *Dunne v. The National Maternity Hospital and Reginald Jackson* [1989] I.R. 91, sets this out as follows:

“Held by the Supreme Court (Finlay C.J., Griffin and Hederman and J.J.) in the appeals in part in directing a retrial and liability and damages in the High Court:

1. That it is well settled that the proper principles applicable to medical negligence were as follows:-

(a) a practitioner was negligent in diagnosis or treatment only if guilty of such failure as no other practitioner of equal specialist or general status and skill would be guilty of acting with ordinary care,

(b) a plaintiff establishes negligence against a medical practitioner by proving his deviation from a general approved practice only upon proving also that the course taken was one which no other medical practitioner of like specialisation and skill would have followed when taking the ordinary care required of a person of his qualifications;

(c) a medical practitioner who establishes that he followed a practice which was general and approved by his colleagues of similar specialisation or skill is nevertheless negligent if the plaintiff thereupon establishes that such practice has inherent defects which ought to be obvious to any person giving the matter due consideration;

(d) an honest difference of opinion between doctors as to which is the better of two ways of treating a patient does not provide a ground for leaving a question to the jury as to whether the defendant who has followed one course, rather than the other, has been negligent;

(e) it is not for a jury (or for a Judge) to decide which of two alternative courses of treatment is in their (or his) opinion preferable but their (or his) function is mainly to decide whether the course of treatment, on the evidence, complied with the careful conduct of a medical practitioner of like specialisation or skill to that professed by the defendant;

(f) where there is an issue of fact, the determination of which is necessary to decide whether a particular medical practice is or is not general and approved, that issue must be left to the jury.

2. That for a practice to be ‘general and approved’, it need not be universal, but must be approved of and adhered to by a substantial number of reputable practitioners holding the relevant specialist or general qualifications. Where certain statements of principle have referred to ‘treatment’ only, these principles must apply in identical fashion to questions of diagnosis.”

3.2 As much of the criticism of what was done by the first named defendant centred around the Data Sheet issued by the manufacturers of Sandostatin LAR a company known as Novartis, and, in particular, what was said to be departures or deviations by the first named defendant from the requirements of this document, it is well to set out the relevant portions of it here.

> *"Clinical particulars*
>
> *Therapeutic indications*
>
> *For symptomatic control and reduction of GH and Somatomedin C plasma level in patients with Acromegaly:-*
>
> *(a) who are controlled on subcutaneous treatment with SANDOSTATIN and*
>
> *(b) who are inadequately controlled by pituitary surgery, Dopamine Agonist treatment, radiotherapy, or in the interim period until radiotherapy becomes fully effective,*
>
> *SANDOSTATIN LAR is indicated for Acromeglic patients for whom surgery is inappropriate.*
>
> *Evidence from studies with S.C. SANDOSTATIN demonstrates that tumour size is reduced in some patients.*
>
> ***Posology and Method of Administration***
>
> *Sandostatin LAR may only be administered by deep, intragluetal injection. The site of repeat intragluteal injections should be alternated between the left and right gluteal muscle (see section Instructions for Use/Handling).*
>
> *In patients who are controlled with the usual therapeutic range of S.C. SANDOSTATIN, it is recommended to start treatment with the administration of 20mg. SANDOSTATIN LAR at four-week intervals for three months without a washout period of S.C. SANDOSTATIN, subsequent dosage adjustment should be based on serum Growth Hormone (GH) and insulin-like growth factor 1/Somatomedine C (IGF1) concentrations and clinical symptoms . . . SANDOSTATIN LAR in patients who are inadequately controlled by pituitary surgery, Dopamine, Agonist treatment, radiotherapy, or, in the interim period until radiotherapy becomes fully effective, a short treatment period of S.C. administration with SANDOSTATIN is recommended to assess the response and systemic tolerability of Octreotide prior to initiating treatment with SANDOSTATIN LAR described above . . .*
>
> *Special Warnings and Precaution for Use*
>
> *. . . Thyroid function should be monitored in patients receiving long-term SANDOSTATIN LAR therapy.*
>
> *Development of gallstones has been reported in ten to twenty per cent of long-term recipients of S.C. SANDOSTATIN. Data recorded on up to nine-month exposure to the new formulation in Acromagalic patients previously treated with S.C. administration of Octreotide, suggest that treatment with SANDOSTATIN LAR does not increase the incidence of gallstone formation as compared to S.C. treatment. However, ultrasonic examination of the gallbladder is recommended before treatment is initiated and at six to twelve month intervals thereafter. If gallstones do occur, they are usually asymptomatic; symptomatic stones should be treated in the normal manner.*
>
> *Hepatic function should be monitored during Octroetide therapy.*
>
> *Octroetide should only be used under hospital specialist supervision, with the appropriate facilities available for diagnosis and evaluation of response . . .*
>
> *Undesirable Effects*
>
> *The main side effects are local and gastrointestinal.*
>
> *Local injection site reactions to SANDOSTATIN LAR may occur, but are usually mild and of short duration. They include local pain and, rarely, swelling and rash. Gastrointestinal side effects include anorexia, nausea, vomiting, abdominal pain, abdominal bloating, flatulence, loose stools, diarrhoea and steatorrhoea. Although measured faecal fat excretion may increase, there is no evidence to date that long-term treatment with Octreotide has led to the nutritional deficiency due to mal-absorption. In rare instances, gastrointestinal side effects may resemble acute intestinal obstruction with progressive abdominal distension, severe epigastric pain, abdominal tenderness and -------*
>
> *Prolonged use of SANDOSTATIN LAR may result in gallstone formation.*
>
> *Because of its inhibitory on insulin reliefs, SANDOSTATIN LAR may impair post-prandial glucose tolerance. In rare instances, with chronic administration, a state of persistent hyperglycaemia may be induced.*
>
> *There have been isolated reports of hepatic dysfunction associated with Octreotide administration . . ."*

3.3 On behalf of the plaintiff, expert evidence was given by Dr. P.D. J. Hardman, a consultant in Clinical Oncology at the James Cooke University Hospital, Middlesbrough, in the United Kingdom, who has a special interest in Radiation Oncology, and by Professor Leslie J. Findlay, a consultant neurologist of the Essex Centre of Neurological Sciences, Queen's Hospital, Romford, Essex. Professor Findlay currently holds and has held a variety of clinical and academic appointments in the United Kingdom and the United States of America.

3.4 On behalf of the defendants, expert evidence was given by Professor S.M. Shalet, who, until 2006, was the head of the Department of Endocrinology at Christie Hospital, Manchester, and the University Hospitals of South Manchester, NHS Trust. Since then, he has been honorary consultant Endocrinologist at the same hospital and Professor of Medicine in Endocrinology at the University of Manchester. His special interest is in the pituitary gland, insofar as adult endocrinology is concerned. Expert evidence was also given on behalf of the defendant by Professor Christopher Thompson, consultant Endocrinologist/Diabetologist at Beaumont Hospital in Dublin.

3.5 Both Dr. Hardman and Professor Findlay appear to have reached a conclusion, or at any rate, a belief, and that part of the first named defendant's reasoning for introducing Sandostatin LAR was a conclusion by the first named defendant that the MRI scan done

on 15th August, 2000, revealed a return or progression of the plaintiff's pituitary tumour. They criticised this state of affairs as having arisen from the failure of the first named defendant to have furnished to the radiologist who conducted the MRI, scan the reports and films from the MRI scan done in 1995 in St. Vincent's Hospital.

3.6 As said earlier in this judgment, and indeed was abundantly clear, the first named defendant did not labour under any such misapprehension and was at all times cognisant of the fact that the plaintiff's tumour, as shown in the August 2000 MRI, had not progressed from that as revealed in the 1995 MRI, but that it was still a very unsatisfactory tumour.

3.7 Indeed, it would appear that Dr. Hardman and Professor Findlay, alone, were misled in this way by the conclusion in the report of the radiographer who carried out the MRI in August 2005.

3.8 Both Professor Findlay and Dr. Hardman criticised the conclusion by the first named defendant that the plaintiff was hyper-secreting Growth Hormone in August 2000. In addition, they both criticised the first named defendant for ignoring the therapeutic indications given in the Data Sheet for Sandostatin LAR on the basis that a single test, namely, the IGF 1, done in August 2001, could not, in isolation, ground a conclusion of hyper-secretion of Growth Hormone, and the plaintiff had no clinical signs of Acromegaly.

3.9 They point to the result of the single Growth Hormone test which was reported on 24th October, 2000, which showed a result of 6.7, well within the normal range, as confirmatory of the fact that the plaintiff was not hyper-secreting Growth Hormone.

3.10 A considerable amount of debate occurred in the evidence as to the nature of the condition known as Acromeghaly. I am satisfied, on the undisputed evidence, that persistent hyper-secretion of Growth Hormone gives rise to clinical Acromeghaly. The condition, in its clinical manifestation, is demonstrated by enlargement of a variety of tissues, most obviously of the head and hands. The tongue can also become enlarged, as also can the heart.

3.11 These clinical manifestations may not be apparent until many years after the condition begins to develop. Of these manifestations, the bone growth associated with enlargement of the head is not reversible when the condition is successfully treated, but soft tissue enlargement is.

3.12 Elevated Growth Hormone levels are associated with development of this condition and also with reduced life expectancy. When elevated Growth Hormone levels are brought back within normal limits, and specifically, when the IGF1 test is brought back to normal, life expectancy returns to normal. Thus, the development of this condition is a serious health hazard and, needless to say, requires treatment, if that is possible.

3.13 Both Dr. Hardman and Professor Findlay were dismissive of the reliance by the first named defendant on the result of a single IGF1 test as indicating hyper-secretion of Growth Hormone. I cannot agree with their opinion in this regard and I prefer the testimony of Professor Shalet and Professor Thompson on this aspect of the case.

3.14 As Endocrinologists, it would seem to me that their expertise in this specific matter must carry much greater weight than that of the plaintiff's experts whose expertise is not specifically in this area of specialisation.

3.15 Professor Shalet's evidence was to the effect that the elevated IGF1 test result was indicative of only one conclusion that he was aware of, namely, hyper-secretion of Growth Hormone. The Growth Hormone result of 6.7 was consistent with that conclusion or, indeed, was consistent with a normal Growth Hormone level. The only result from a Growth Hormone test which would have been inconsistent with the IGF1 result would have been a Growth Hormone test result of less than one. This, he said is because Growth Hormone levels fluctuate throughout the day and are affected by a variety of human stress experiences.

3.16 Growth Hormone is released normally in pulses, mainly at nightime. A typical day time Growth Hormone test result for someone without elevated Growth Hormone will be less than one, but a random test result maybe much higher if it catches the episodic release of Growth Hormone.

3.17 The normal Glucose Tolerance Test to ascertain hormone levels are done by taking samples at different times of the day to give an effective average. In this case, the test was done in August 2000, but due to error, the full test was not reported and only one growth level was reported, namely, the 6.7 result.

3.18 The IGF1 test measures a much more consistent release of Growth Hormone and is seen by Endocrinologists as a much more reliable indicator on its own of the presence of hyper-secretion. The evidence of Professor Thompson was to the effect that 30% of Endocrinologists will rely solely on the IGF1 test as an indication of hyper-secretion of Growth Hormone and will commence treatment on Sandostatin LAR or a similar drug, on that basis alone, and in particular, his evidence was that in the United States, there is greater reliance amongst Endocrinologists on the validity of the IGF1 test alone as an indicator of elevated Growth Hormones.

3.19 In addition, both Professor Shalet and Professor Thompson stressed the plaintiff's previous history, and in particular, the fact that hyper-secretion of growth hormone had been noted in the mid-1990s when under the care of Dr. Mitchell. At that stage, it was established that the plaintiff's pituitary tumour was uniquely rare, in that it secreted excessively Prolactin, THS and Growth Hormone.

3.20 Thus, these witnesses were of the opinion that in light of the plaintiff's history, together with the results of the IGF1 test in August 2000, the first named defendant was right to conclude that the plaintiff was hyper-secreting Growth Hormone and, thus, was biochemically Acromegalic.

3.21 In addition, Professor Thompson expressed the opinion that she was probably clinically Acromegalic at that time as well, although the first named defendant, or indeed, Professor Shalet, when he examined her on 8th February, 2010, did not find any clinical signs of Acromegaly.

3.22 Both Professor Shalet and Professor Thompson expressed concern at the Growth Hormone test result of 6.7 and doubted whether the fact that it was within the normal range could support comfortably a conclusion that it did not indicate elevated Growth Hormone.

3.23 The second named defendant was of a similar view, and a factor which impinged upon the consideration of whether that figure of 6.7 represented normal Growth Hormone levels or not, was the fact that the plaintiff was on high doses of Bromocryptine which can suppress Growth Hormone levels, although in a minority of patients. The plaintiff, in this case, had, for a very long time, been on a very high dose of Bromocryptine.

3.24 Professor Thompson gave evidence to the effect that the vast majority of Endocrinologists would prescribe Sandostatin LAR on the basis of an elevated IGF1 test and a single Growth Hormone test revealing a result of greater than five. He estimated approximately ninety per cent of Endocrinologists, based on responses to debates at conferences as being of this view, and, similarly, that thirty per cent of Endocrinologists proceeded on the basis of the IGF1 test alone.

3.25 Although Professor Shalet did not indicate how he saw the balance of opinion on this topic amongst consultant Endocrinologists, he was emphatic that he himself, in the light of her history, and the test results in August 2000,would have considered the plaintiff as an appropriate patient for the administration of Sandostatin LAR.

3.26 I accept the evidence of Professor Shalet and Professor Thompson on this question and I am satisfied that the second named defendant was correct in prescribing Sandostatin on the basis that the plaintiff was hyper-secreting Growth Hormone in August 2000, and was, at the very least, biochemically Acromegalic.

3.27 This brings me to the next disputed topic and that is compliance with the requirements of the Data Sheet.

3.28 The second named defendant considered that so far as therapeutic indications were concerned, the plaintiff's case fell to be considered under clause (b) quoted above.

3.29 In the first place, I am satisfied that the plaintiff, was, on the basis of her elevated Growth Hormone, rightly considered as a patient with Acromegaly. given that she had a pituitary tumour which had a history of hyper-secretion of Growth Hormone and she was, in August 2000, hyper-secreting Growth Hormone, it was apparent that she was not adequately controlled by her pituitary surgery, or by Dopamine Agonist treatment i.e. her Bromocryptine, or by the radiotherapy that had been done in 1994. Whilst it might very well have been the case that in due course the radiotherapy might have kicked in to control excessive levels of Growth Hormone and other hormones, so far as Growth Hormone was concerned, in August 2000, radiotherapy had not become effective.

3.30 Happily for the plaintiff, in the years subsequent to 2001, the beneficial effect of the radiotherapy in this regard does appear to have become effective, but as of August 2000, her Growth Hormone was evidently unacceptably high and I am satisfied that the first named defendant was right to conclude that a therapeutic intervention was required. Before passing from this topic I should draw attention to part of the evidence of Professor Thompson who felt that if anything, the first named defendant was excessively cautious in waiting until September, 2000 to put the plaintiff on Sandostatin or a similar drug.

3.31 Professor Thompson was of the opinion that apart from the problem with the plaintiff's growth hormone the prospect of otherwise controlling the tumour and T.S.H levels, by the use of this type drug warranted its prescription for the plaintiff, and he said that nearly all of his patients with pituitary tumours were on this type of drug.

3.32 Thus far, there is no doubt, in my view, that the first named defendant complied with the therapeutic indications in the Data Sheet.

3.33 The next disputed question was the selection of Sandostatin LAR as distinct from Sandostatin S.C. Professor Findlay and Dr. Hardman strongly criticised the first named defendant for not having commenced treatment by way of the subcutaneous injection given three times daily for a short period as recommended in the Data Sheet. They expressed the opinion that this should be done in order to ascertain the tolerance of the plaintiff to the drug and also to test her response to the drug.

3.34 The first named defendant explained the selection of the Sandostatin LAR as distinct from Sandostatin S.C. as being normal practice in Endocrinology on the basis that the Sandostatin S.C. as a testing ground with a view to leading on to Sandostatin LAR is not a reliable or good predictor of either response or tolerance levels.

3.35 In this regard, Professor Shalet and Professor Thompson agreed with the first named defendant's position. Both of these expert witnesses explained that so far as tolerance is concerned, only about three per cent of patients who have been put on Sandostatin S.C. decline to go ahead with Sandostatin LAR because of the side effects which are largely gastrointestinal upset and, secondly, so far as response is concerned, the Sandostatin S.C., whilst it can indicate a good response, a poor response is not a reliable predictor of a good or bad response on the Sandostatin LAR, namely, the long acting version. Hence, patients can be deprived of the benefit of Sandostatin LAR, when they might benefit from it, because of a misleading poor response to the subcutaneous version.

3.36 The first named defendant additionally explained that so far as tolerance was concerned, that whilst the long acting drug over one month can administer a heavy load of the active ingredient, because it released in a very even way over a four-week period, or even longer, the short-term or immediate load can be considerably less than the load administered by the subcutaneous injections given three times a day.

3.37 Thus, the impact of the Sandostatin LAR may not be as acute in terms of generating unpleasant gastrointestinal side effects as the subcutaneous version.

3.38 Professor Shalet's evidence was that because of the foregoing reasons, fifty per cent of Endocrinologists in the United Kingdom do not use the subcutaneous version of Sandostatin but introduce the Sandostatin LAR at the outset of treatment. Professor Thomspson's evidence was that in his centre in Beaumont Hospital which treats a very large number of patients with pituitary problems, the prevailing practise is to not use Sandostatin subcutaneous but to move straightway to the long acting drug. He explained that the more modern version of these drugs now do not have a subcutaneous version.

3.39 Professor Findlay and Dr. Hardman accepted in cross-examination that neither of them had ever prescribed the drug in question in this case and I am quite satisfied that I should prefer the evidence of the first defendant and Professor Shalet and Professor Thompson on the issue of the selection of Sandostatin LAR as opposed to Sandostatin S.C.

3.40 This brings me to the question of monitoring of the plaintiff after the commencement of the Sandostatin LAR treatment. Criticism of the manner in which the first named defendant monitored the plaintiff was made by Professor Findlay and Dr. Hardman.

3.41 The Data Sheet identifies a number of risks or side effects which call for monitoring during treatment. The first mentioned is thyroid function which the Data Sheet says should be monitored in patients receiving long term Sandostatin LAR. Secondly, there is a risk of the formation of gallstones which occurs in 10 to 20% of long term recipients of Sandostatin, be it of the S.C. variety or the LAR version. The Data Sheet recommends an ultrasonic examination before treatment is commenced and at six to twelve month intervals thereafter. It goes on to say that if gallstones do occur they are usually asymptomatic and this was borne out by the evidence of Professor Thompson of his experience of the occurrence of gallstones in these circumstances. The Data Sheet also

recommends the monitoring of hepatic function, because of isolated reports of hepatic dysfunction associated with octreotide administration.

3.42 Apart from these side effects, monitoring would be necessary to establish the patient's response to the drug to see whether it was being effective. The Data Sheet is silent on this topic, apart from recommending a short period of treatment on Sandostatin S.C. to assess response and tolerability. All of the expert evidence and, indeed, that of the first defendant, recognised the need for monitoring the patient's response to the drug. The issue here is when this should be done. The first defendant has planned to do this approximately four months after the plaintiff had been on the treatment, which would have been approximately mid-January, 2001. The plaintiff's experts and in particular Professor Findlay stressed three months as the appropriate time. They also criticised the failure to have carried out an ultrasound to establish a base line for gallstones before the treatment commenced.

3.43 In the event, as a result of the plaintiff being admitted to hospital on the 2nd January, 2001, much of the monitoring recommended was carried out. The plaintiff had routine blood tests done which revealed her hypothyroidism. I infer that these routine blood tests would also have covered her hepatic function. During this visit also an abdominal ultrasound was carried out on the plaintiff to test for gallstones, but happily for the plaintiff she had not developed these. The only thing that was not done when the plaintiff was in hospital from the 2nd to the 4th January, was that she did not have tests to determine the effect of the drug on her growth hormone levels, namely, an IGF 1 test and a Glucose Tolerance Test. Two reasons emerged for this not having been done. The first of these was that because of the reaction of the plaintiff to the first named defendant and her insistence on discharging herself, it would have been very difficult, if not wholly impracticable, to have carried out the Glucose Tolerance Test in the time available and in the fractious circumstances prevailing. The second reason, and this was stressed by Professor Shalet, was that the presence of hypothyroidism, uncorrected, would have skewed or distorted the results of these tests and it was better to postpone these until her hypothyroidism was corrected. As against this latter consideration, Professor Thompson's evidence was, that whilst the hypothyroidism would have distorted the results of these tests, nonetheless they could have been done with due allowance being made for the effect on them of the hypothyroidism. Professor Thompson also favoured a shorter monitoring period, saying that in his centre monitoring for the efficacy of the drug is done at six to eight weeks by which time meaningful results can be obtained from these tests.

3.44 I am satisfied that most of the recommended monitoring was carried out within an appropriate time frame by the first named defendant. I am quite satisfied that it is not standard practice in endocrinology to carry out an ultrasound test before commencement of treatment, notwithstanding what is said in the Data Sheet, The reverse appears to be the established practise, which of course makes eminent sense in light of the fact that even if gallstones do occur they are usually asymptopmatic. It could be said that the recommendation for a pre-treatment Ultra Sound in the Data Sheet, is or was, an abundance if not an excess of caution.

3.45 The postponement of testing growth hormone levels from January to a later date was clearly the subject matter of a difference of opinion amongst the experts. It would appear to me that good grounds existed upon which the first named defendant was justified in postponing these tests. Firstly, there were the unfortunate circumstances prevailing during the plaintiff's admission to hospital in January which would have made it very difficult to have completed the Glucose Tolerance Test. Secondly, the compromise of the test by uncorrected hypothyroidism was a significant factor. Thirdly, the fact that the injection Sandostatin LAR which should have been given at the end of December or beginning of January was not given, meant that testing during January would not have yielded a reliable result and until the effect of the resumption of treatment with the next injection could be properly assessed, there would have been little point in doing these tests until after the last two injections in February and March had taken effect.

3.46 The first named defendant at this stage still hoped to persuade the plaintiff to continue with the drug on a long term basis, but as events transpired the consultation on the 20th February, 2001, in effect marked the end of their professional relationship, although the plaintiff did take the last of the six injections at the beginning of March, 2001.

3.47 I cannot see that there was any failure on the part of the first named defendant in his duty of care to the plaintiff in the monitoring of the plaintiff after the treatment with Sandostatin LAR commenced in September, 2000.

3.48 This brings me to a consideration of the advice given by the first named defendant to the plaintiff in September, 2000 before the commencement of her treatment on Sandostatin LAR. As said earlier, the plaintiff's evidence was that she was given an impression of her tumour which conveyed to her that it was growing and progressing and that unless she went on the treatment recommended her health would be in grave peril. She also said that she was told nothing about the side effects of the drug. The evidence of the second named defendant was that he positively recommended the Sandostatin LAR and that he presented the results of the plaintiff's test to her in a very positive manner, that her elevated growth hormone levels provided an opportunity to use the Sandostatin LAR which was proven to be successful in reducing growth hormone levels and might reduce the size of her tumour. I accept the first named defendant's evidence that he did tell her of the likelihood of gastrointestinal upset including vomiting and diarrhoea and probably indicated that these would be transient. There is not doubt he did not mention the risk of developing gallstones or of any interference with hepatic function or developing hypothyroidism. Whilst this all took place in the course of a telephone conversation, a far from ideal forum of communication in the circumstances, nonetheless in the context of a professional relationship which had been ongoing for five years, at that time, and having regard to the fact that the plaintiff had been telephoning to obtain the results of her tests, and as soon as they became available the first defendant telephoned her, I would not accept that the first named defendant's conduct in dealing with the plaintiff over the phone in this way, constituted a failure on his part to observe a standard of care appropriate to his status and specialisation. Soon afterwards, in response to the plaintiff's gastrointestinal problems which she communicated directly to the first defendant by telephone, a consultation was held on the 18th September, 2000. There is no record of what was discussed at that consultation, but I am quite satisfied that in the course of it, the first defendant did encourage the plaintiff to continue with the Sandostatin LAR and discussed with the plaintiff the gastrointestinal distress which she was clearly up to then suffering, and, as a consequence of which the first defendant reduced the dose from 20mgs to 10mgs for the ensuing injections.

3.49 Notwithstanding the fact that the plaintiff's tumour was stable, there is no doubt that it continued to have the potential to do very great harm to the plaintiff' health and I accept the first named defendant's contention that it would have been unacceptable to do nothing if a treatment option was available. The availability of Sandostatin in a long acting format and in the context of elevated growth hormone levels clearly did create a treatment opportunity which had not existed prior to the late 1990s. Sandostatin S.C. had been considered at an earlier time as mentioned above, but rejected as unacceptable to the plaintiff. In view of the success rate of 60 to 65%, of Sandostatin in reducing excess growth hormone levels, the first defendant was not only entitled but in my view professionally obligated to have presented this treatment to the plaintiff and to have offered positive encouragement to her to undergo it. I am satisfied he did tell her of the main side effect of the drug, namely gastrointestinal upset. He could not have told her about the risk of developing hypothyroidism as a side effect because that was not known then. The risk of interference with hepatic function appears to have been remote and according to the Data Sheet based only on isolated reports. In my opinion, it could not be said that he failed in his professional duty of care to her in not mentioning this. Although the risk of developing gallstones was significant, it would appear from the Data Sheet that this was related to long term use of Sandostatin LAR and occurred in only 10-

20% of patients, and if it did occur, was likely to be a asymptomatic. It could not be said, in my view, that his failure to mention this amounted to a breach of his duty of care to the plaintiff in the context of proper encouragement to a reluctant or treatment adverse patient to undergo a treatment which had a very good prospect of rendering much safer a potentially serious risk to her health and life expectancy.

3.50 As events turned out, the first defendant's encouragement of the plaintiff to undergo this treatment would appear to have been vindicated. The tests carried out by Dr. Howel Walsh in April and May of 2001, and particularly the IGF1 test, indicated that the plaintiff's Growth Hormone level was reduced to well within the normal range and very significantly below the level shown in the August, 2000 IGF1 test. An MRI carried out in November, 2001, showed a ten per cent reduction in the size of the plaintiff's tumour. The weight of the evidence points to these beneficial outcomes being the probable result of the six Sandostatin LAR injections from September to March, 2001.

3.51 Undoubtedly, the continuing stability of her condition thereafter, and in particular, a further reduction in the size of her tumour of approximately 50% recorded in an MRI Scan conducted in 2005, are to be attributed to the long term effects of the radiotherapy taking effect but pending that happening the Sandostatin LAR was a therapeutic option, which in my opinion was correctly and appropriately offered to the plaintiff.

3.52 The first named defendant was criticised for not offering other alternatives such as further radiotherapy or surgery and not referring the plaintiff to these specialities.

3.53 This criticism is to my mind somewhat speculative. Without doubt, the first named defendant was fully aware of what surgery and radiotherapy the plaintiff had received and the extent of any actual or potential benefit and risks from these. I am quite satisfied that it was apparent to the first defendant, as it would have been to any other endocrinologist of his status, that there was little or no prospect of any treatment that could be recommended to, or might be acceptable to the plaintiff, arising from those disciplines. To my mind, there is an element of artificiality in these criticisms. The treatment of the plaintiff with the Sandostatin LAR had an established prospect to benefit the plaintiff with the risk or side effects of a relatively minor character. It is hard to comprehend the suggestion that surgery or further radiotherapy would be preferred or could conceivably be recommended to the plaintiff at the relevant time, when there was available a drug therapy with relatively minor risks attached.

3.54 I am satisfied that the first named defendant did not breach his duty of care to the plaintiff in the information and advice given by him to the plaintiff prior to and during her treatment with Sandostatin LAR.

3.55 The first defendant was also criticised for delegating the administration of the injections to the plaintiff's general practitioner, Dr. Len Harty. It was suggested that this was in breach of the guidelines in the Data Sheet. The relevant portion of the Data Sheet reads as follows:

> *"Octreotide should only be used under hospital specialist supervision with the appropriate facilities available for diagnosis and evaluation of response."*

3.56 Manifestly, in this case, the Sandostatin LAR was used under the supervision of a hospital consultant, namely the first named defendant and there was available in the second named defendant's hospital appropriate facilities available for diagnosis and evaluation of response. If the injections had been given to the plaintiff in hospital they would have been administered by a nurse. It seems odd, at the very least, to suggest that an experienced general practitioner was not competent to administer these injections.

3.57 I am quite satisfied that there is no merit whatsoever in this criticism. There was an obvious benefit in having these injections administered by the plaintiff's general practitioner. Firstly, it was undoubtedly much more convenient for the plaintiff. Secondly, the general practitioner could be involved in monitoring or observing how the patient with whom he was very familiar, responded to the treatment and could initiate speedy action if that was required as, in fact, occurred. It was her general practitioner who referred her to hospital at the beginning of January, 2001.

4. Consent

4.1 This brings me finally to the issue of informed consent. The question of informed consent was considered in detail by Kearns J. in the case of *Geoghegan v. Harri*s [2000] I.R. 536. The head note to the case summarises the conclusions of the court as follows:

> *"Held by the High Court (Kearns J.)*
>
> *1. That the defendant was obliged to give a warning to the plaintiff of any material risk which was a known or foreseeable complication of an operation. Despite the fact that the nature of the risk in this case was extremely remote it was a known complication and a warning of the risk was required. (Walsh v. Family Planning Services Ltd [1992] 1 I.R. 496 applied.*
>
> *2. That the test to be adopted by the court, as to what risks ought to be disclosed to a patient before an operation, was the test of the reasonable patient. By adopting that test it was the patient, thus informed, rather than the doctor, who made the real choice as to whether the treatment was to be carried out. Walsh v. Family Law Planning Services [1992] 1 I.R. 496 considered.*
>
> *3. That, when deciding whether or not a warning would cause a patient to forego an operation, the court was first to adopt an objective test. That test was to yield to a subjective test where there was clear evidence in existence from which a court could reliably infer what a particular patient would have decided.*
>
> *4. That no category of inquisitive patient existed in Irish law because of the onerous obligations imposed on the medical provision to warn patients of all risks with severe consequences regardless of their infrequency."*

At p. 549 of the report Kearns J. said the following:

> *"The application of the reasonable patient to test seems more logical in respect of disclosure. This would establish the proposition that, as a general principle, the patient has a right to know and the practitioner a duty to advise of all material risks associated with the proposed form of treatment. The court must ultimately decide what is material. "Materiality" includes considerations of both (a) the severity of the consequences and (b) the statistical frequency of the risk. That both are critical is obvious because a risk may have serious consequences and yet historically or*

predictably be so rare as not be to be regarded as insignificant by many people. For example, a tourist might be deterred from visiting a country where there had been an earthquake causing loss of life, but if told the event happened fifty years ago without repetition since, he might well wonder why his travel agent caused him unnecessary worry by mentioning it at all.

The reasonable man, entitled as he must be to full information of material risk, does not have impossible expectations nor does he seek to impose impossible standards. He does not invoke only the wisdom of hindsight if things go wrong. He must be taking as needing medical practitioners to deliver on their medical expertise without excessive restraint or gross limitation on their ability to do so.

The decision in Walsh v. Family Planning Services Ltd [1992] 1 I.R. 496 effectively confines the test of materiality to severity of consequences only. This approach is best capsulated in the memorable passage of McCarthy J. when he stated at p. 521:

'…those concerned…if they knew of such a risk, however remote, had a duty to inform those who are critically concerned with that risk. Remote percentages of risk loss their significance to those unfortunate enough to be 100% involved.'

However, the attractiveness of the observation should not occlude the possibility that at times a risk may become so remote, in relation at any rate to the less and most serious consequences, that a reasonable man may not regard it as material or significant. While such cases may be few in number, they do suggest that an absolute requirement for disclosure in every case is unduly onerous, and perhaps in the end counter productive if it needlessly deters patients from undergoing operations which are in their best interests to have.

As pointed out by Mr. John Healy in his book "Medical Negligence Common Law Perspectives" at p. 99:

'Materiality is not a static concept.'

If the assessment of materiality is to "abide a rule of reason" any absolute requirement which ignores frequency seems at variance with any such rule.

Each case it seems to me should be considered in the light of its own particular facts, evidence and circumstances to see if the reasonable patient in the plaintiff's position would have required a warning of the particular risk."

Further, on in his judgment at p. 559 Kearns J. says the following:

"A reasonable patient would then place in the balance of making any decision the benefits associated with the procedure....

Commencing with the objective test, it seems to me that had a proper warning been given by the defendant to a reasonable patient in the plaintiff's position such a reasonable patient was more likely, for the reasons stated, to have proceeded with the operation. However, as a credible and reliable picture emerges overall and analysing the evidence particular to this case, the issue can and must be resolved by reference to the subjective test of what the plaintiff himself can as a matter of probability would have done…"

Earlier in the judgment at p. 550 under the heading "Causation" Kearns J. said the following:

"It is not sufficient to establish that a warning should have been given but was not given to entitle the plaintiff to recover damages. They must also establish that that, had he been given a proper warning, he would have opted to forego the procedure…"

4.2 In the later case of *Fitzpatrick v. White* [2008] 3 I.R. 551, the Supreme Court held in following the judgment of Kearns J. in *Geoghegan v. Harris* in that if there was a material risk inherent in an operation which would affect the judgment of a reasonable patient, then in the normal course it was the responsibility of a doctor to inform the patient of that material risk, and that a risk was material if, in the circumstances of the particular case, a reasonable person in the patient's position, if warned to the risk, would be likely to attach significance to it.

4.3 In this case Dr. White on behalf of the plaintiff urged me to follow the judgment of the House of Lords in the case of Chester v. Afshar [2004] UKHL 41, in determining the causation linkage between the injuries of which the plaintiff complains and the deficiencies claimed in the information and advice given by the first defendant to the plaintiff before commencement on the Sandostatin LAR treatment. The relevant aspect of the decision of the House of Lords in this case as set out in the head note reads as follows:

"(1) That, since the judge had not found that, if properly informed, the claimant would never have undergone the operation and since the risk which eventuated was liable to occur at random irrespective of the skill and care with which the operation might be performed, the defendant's failure to warn neither affected the risk nor was the effective cause of the injury she sustained, so that, applying conventional principles she could not satisfy the test of causation…

(2) But that, dismissing the appeal (Lord Bingham of Cornhill and Lord Hoffmann dissenting), the issue of causation was to be addressed by reference to the scope of the doctor's duty, namely, to advise his patient of the disadvantages or dangers of the treatment he proposed; that such a duty was totally connected with the need for the patient's consent and was central to her right to exercise an informed choice as to whether and, if so, when and from whom, to receive treatment; that since the injury she sustained was within the scope of the defendant's duty to warn and was the result of the risk of which she was entitled to be warned when he obtained her consent to the operation in which it occurred, the injury was to be regarded as having been caused by the defendant's breach of that duty; and that, accordingly, justice required a narrow modification of traditional causation principles to vindicate the claimant's right of choice and to provide a remedy for the breach . . . "

4.4 As will be apparent, any differences between this statement of the law and law on causation in this jurisdiction , as stated by Kearns J. in *Geoghegan v. Harris*, is immaterial in so far as the facts of this case are concerned, hence, it is unnecessary for me to express any opinion on that question.

4.5 The plaintiff, in her evidence, as already discussed, raised a number of matters which were characterised as misrepresentations to her concerning the state of her pituitary tumour in August 2000. Principal among these concerned whether or not her tumour was growing and progressing as of that time. The view that it was so growing and progressing came to be characterised as misrepresentation by the first named defendant as a result of, it would seem to me, a combination of two circumstances, both unfortunate.

4.6 In the first place, the plaintiff, notwithstanding her medical history over the previous eighteen years, had a belief that her tumour had been substantially removed, leaving only something, as she described it, the size of her fingertip, and was thus shocked and alarmed when confronted with the description of its actual size in September 2000.

4.7 The other circumstance was the erroneous conclusion that the first named defendant had concluded that the tumour had progressed and grown because of the failure of to supply the radiologist with the earlier MRI film and report and because of the conclusion stated in the report on the August 2000 MRI. As said earlier, it is clear from the evidence that the first named defendant had not reached any such conclusion. However, it is quite clear that the impression that the plaintiff took from the telephone conversation on 5th September, 2000, was one in which she thought her condition had deteriorated alarmingly, that her heath was in grave peril.

4.8 I do not think that the first named defendant can be blamed for this impression because I am satisfied that the information he relayed to her concerning her tumour was accurate and appropriate, both as to the size and mass of the tumour, and its hyper-secretion of Growth Hormones.

4.9 It is unfortunate that the plaintiff's state of mind at the time was such that this information affected her in the way it did, but in my opinion, the first named defendant did no more and no less than was his duty, namely, to convey full and accurate information to her concerning her condition.

4.10 There is no doubt that the first named defendant introduced the drug, Sandostatin LAR to her in positive terms, and in general, was positive or upbeat about the opportunity for therapeutic benefit that it presented. It is quite clear that he did assure the plaintiff of a good prospect of success from the drug, which was entirely justified, having regard to its proven success rate, but he did not exaggerate its potential benefit. Because of the plaintiff's state of mind at the time, she obviously perceived the positiveness or cheerfulness of the first named defendant's presentation as flippant. The real problem that emerged in the professional relationship, stemmed not from anything inappropriate said or omitted, by the first named defendant, but from the state of mind of the plaintiff at that time concerning her condition.

4.11 Insofar as the plaintiff came away from that conversation on 5th September, 2000, believing that if she did not take the Sandostatin LAR, she would be, as she put it herself, "*in big trouble*", I am satisfied that her conclusion in that regard was not caused or contributed to by any inappropriate advice or information given by the first named defendant or any essential information withheld concerning the up to date of the state of the plaintiff's pituitary tumour or the appropriateness of the Sandostatin LAR treatment. Insofar as the description by the first named defendant of the plaintiff's condition and the presentation by him of the proposed treatment was concerned, he did all that was necessary to procure from the plaintiff an informed consent to the treatment. If one takes the plaintiff's state of mind as it was for the purpose of considering the consent issue, this conclusion could be no different because the first named defendant was not aware, I am satisfied, of the plaintiff's state of knowledge concerning her tumour and could not be blamed for the deficiencies in her knowledge of her condition.

4.12 This brings me to the question of the risks or side effects associated with Sandostatin LAR and whether the advise, information or warnings concerning these, given by the first named defendant were adequate.

4.13 Quite clearly, the principal side effect from this drug is that of gastrointestinal upset involving vomiting and diarrhoea. I am quite satisfied that the first named defendant did discuss this with the plaintiff before treatment initiated and in the consultation which occurred on 18th September, 2000, which came about because of the plaintiff's experience of gastric upset, I have no doubt this problem must have been discussed.

4.14 Thus, with clear knowledge of this side effect, the plaintiff undertook the treatment and continued with it and, indeed, completed the treatment by taking the last two injections at the beginning of February and March 2001, clearly knowing of and having experienced this side effect. If she felt impelled to take that course by a belief that her health was in imminent peril because of the information conveyed to her by the first defendant, notwithstanding her revulsion to the treatment and it's disclosed side effects, as said before, that outcome was the result of her surprising if not extraordinary lack of awareness of the true state of her tumour, at that time, in respect of which there is no evidence which would support fixing the blame for this on the first named defendant.

4.15 Next in prominence as a known side effect was the development of gallstones.

The first named defendant acknowledged that he did not warn the plaintiff of the risk of this occurring. The risk involved here is that in between ten and twenty per cent of patients who are long-term on Sandostatin, gallstones do occur, but when they do, they tend to be asymptomatic, a view shared and explained by Professor Thompson in his evidence.

4.16 In my opinion, for the purposes of warnings to a patient in advance of this treatment, the risk of the formation of gallstones in these circumstances could not be considered to be of such seriousness or severity as to be considered a "*material*" risk and therefore, in my view, the first named defendant was not obliged to warn the plaintiff of this risk before treatment commenced in order to obtain an informed consent.

4.17 In the events that occurred, of course, the plaintiff did have an Ultrasound for gallstones carried out when in hospital in early January 2001, and she had not developed gallstones. Thus, the plaintiff has no grounds for complaint in this regard.

4.18 The Data Sheet also mentions hepatic dysfunction, but only of isolated reports of this. No warning was given in respect of this, and as the plaintiff did not develop any such dysfunction, there are no grounds for complaint here.

4.19 Finally, the Data Sheet mentions thyroid function and recommends monitoring in patients receiving long-term Sandostatin LAR therapy. The plaintiff did develop hypothyroidism as a side effect of the Sandostatin LAR. I accept the first named defendant's evidence that at the time the drug was administered, hypothyroidism was not a known side effect of the drug and, consequently, could not have been warned prior to the plaintiff's treatment.

4.20 I am satisfied, therefore, that there was no failure on the part of the first named defendant to give appropriate advice or warnings to the plaintiff concerning harmful risks or side effects from her treatment with Sandostatin LAR such as to vitiate the consent given by the plaintiff to that treatment.

4.21 In light of this conclusion, it is unnecessary for me to consider the submission made by Dr. White to the effect that a different test for causation be applied to that set out in the judgment of Kearns J. in *Geoghegan v. Harris*, as discussed above, and that I should adopt the test considered in the judgments of the House of Lords in *Chester v. Afshar*.

4.22 It would appear that the plaintiff came to believe that the first named defendant was experimenting on her with the Sandostatin LAR drug, in effect, using her as a guinea pig. Notwithstanding the fact that there was no basis whatever for this belief, it greatly affected her, and to a very large extent, led to her loss of confidence in the first named defendant. As this is mentioned in the pleadings and came up in evidence, it should be said that there was no evidence to sustain the plaintiff's belief in this regard. On the contrary, the evidence clearly established that the first named defendant had no connection whatsoever with this drug apart from using it in the course of clinical practice and had not been involved in any clinical trials with this drug and had no connection to its manufacturer, Novartis.

5. Conclusion

5.1 I am satisfied that there was no negligence on the part of the defendants in any aspect of the treatment of the plaintiff with the Sandostatin LAR drug. I am also satisfied that the plaintiff's consent to that treatment was a valid informed consent.