

Swedish & Norwegian Testicular Cancer Projekt, SWENOTECA

Onkologiskt centrum, Lund, Sweden

Kontor for klinisk kreftforskning, Bergen, Norway

SWENOTECA VII

A revised continuation of SWENOTECA V

TESTICULAR SEMINOMA

CLINICAL STAGE I – SURVEILLANCE AND TREATMENT

CLINICAL STAGE IIA – RADIOTHERAPY

ADVANCED AND RELAPSED DISEASE

Not Valid



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Information to be given to the patient at completed follow-up – to be completed during 2007, see <http://www.ocsyd.lu.se>

PREFACE

The Swedish & Norwegian Testicular Cancer Project, the SWENOTECA, started in 1981 with programs for staging, treatment and follow-up of non-seminomatous germ cell cancer.

The first program for testicular seminoma, SWENOTECA V, was introduced in 2000.

This is the second seminoma program, SWENOTECA VII, introducing risk adapted adjuvant treatment for localized seminoma.

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Flow-sheet

Seminomatous Germ Cell Testicular Cancer, SWENOTECA VII

Testicular tumour

Ultrasound both testicles

Physical examination

History of prior testicular disorders and hereditary information



Tumour markers: AFP, β -HCG, LDH, PLAP (optional)

Hormone levels: Testosterone, LH, FSH, SHBG

Spermcount & cryopreservation (recommended)

Inguinal orchiectomy and biopsy contralateral testicle



CIS → See addendum 24

Seminomatous testicular cancer (seminoma)

AFP normal (<1.5 normal range, repeated and stable values)

Risk factors: **Rete testis invasion**

Size of tumour > 4 cm



Clinical staging procedure

Tumour markers post-op: β -HCG, AFP, LDH (if AFP)

Spermcount & cryopreservation, (consider if not done earlier).

Chest X-ray, CT scan of thorax, abdomen and pelvis

TNM-classification



Treatment

Clinical stage I disease (CS I)

0–1 risk factor → **surveillance** or carboplatin x 1

2 risk factors → **carboplatin x 1** or surveillance

Option: radiotherapy to para-aortic nodes 2 Gy x 10 to 20 Gy. (For patients with previous inguinal or scrotal surgery, or T4 tumour, see addendum 23 Radiotherapy)

Clinical stage IIA disease (CSIIA)

Radiotherapy 1.8 Gy x 15 to 27 Gy to para-aortic and ipsilateral iliac nodes, or 4 x EP chemotherapy

Clinical stage $\geq$ IIB (CS IIB–IV)

4 courses of EP-chemotherapy. If contraindications to chemotherapy or small tumour volume, treatment with radiotherapy to para-aortic and ipsilateral iliac nodes 1.8 Gy x 20 to 36 Gy might be an option in stage IIB. In very advanced disease BEP chemotherapy should be used.

Send in the SWENOTECA registration form to the national secretariat. After treatment, send in the treatment form. Follow-up according to schedule, see addendum 25. Send in FU forms after each follow-up.

In case of relapse – new full staging. Histological verification of relapse if possible.

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1 Background

1.1 General information

Testicular tumours accounts for about 1% of all male malignancies and are in 95% of germ cell origin. (1) Seminoma constitutes approximately 50% of all germ cell tumours and is increasing in incidence. (2) The peak incidence occurs between thirty and forty years of age. Data from the Swedish and Norwegian cancer registries show an increase in seminoma from about 90 cases annually in Sweden (1983-1987) to about 150 (2004) and from about 75 cases annually in Norway (1983–1987) to about 150 (2004).

Little is known about etiological risk factors for the development of testicular tumours. Ten percent of the patients have had a history of cryptorchidism. Some epidemiological studies show a significantly increased percentage of pure seminoma as compared to germ cell tumours of other histologies in men with undescended testis. (3) Other risk factors are hypotrophic testicle and infertility. (4)

At the time of diagnosis, about 85% of the patients will be in clinical stage I and IIA. Due to a high radiosensitivity, seminoma has for decades been documented to be curable by radiotherapy in early stages. A surveillance policy for clinical stage I and treatment only in the case of relapse has in observational studies shown the same total cure rate of about 98%. (5) In a recent large randomised study of 1447 pts in stage I (no T4 tumours), with a median follow up of 4 years, comparing one course of adjuvant carboplatin chemotherapy with adjuvant radiotherapy, outcome was found to be similar with a relapse rate of 3–4% and no serious acute side effects. (6) The pattern of relapse differed with more abdominal retroperitoneal relapses after carboplatin chemotherapy compared to those treated with radiotherapy who experienced more relapses outside the retroperitoneal field. The development of highly effective chemotherapy has improved the curability also for advanced disease, and the total cure rate is now above 95%.

1.2 International guidelines for the treatment of CSI

Recently the European Germ Cell Cancer Consensus Group (EGCCCG) as well as the EAU-guidelines group list surveillance, carboplatin and radiotherapy as equivalent treatment alternatives. (7, 8)

1.3 SWENOTECA experience

During April 2000-Nov 2005, about 850 patients with seminoma in all stages have been included in the SWENOTECA V program. Median follow-up time is about 37 months (4–67). Median age at orchiectomy was 36 years. Median size of the testicular tumours was 3.6 cm (1.2–8.7). About 87% of the patients were in stage I. Patients in clinical stage I had the choice between adjuvant radiotherapy or surveillance. In Norway 40% of the 201 patients were treated with adjuvant radiation therapy versus 62% of the 475 Swedish patients. Recurrence in clinical stage I patients were seen in 13 patients on surveillance in Norway, and 1 patient in the radiation group, i.e., 10.7% and 1.3%, respectively; and in 13 (7.3%) of the Swedish patients on surveillance, and in 2 patients treated with radiation therapy (0.7%). Almost all recurrences were in the paraaortic lymph nodes. Median time to relapse was 10 months (range 3–44 months).

1.4 Follow up

Follow up is important, especially in surveillance patients. It is equally important in patients treated with carboplatin until data on long-term follow-up emerge. More than 80% of the relapses both for stage I and II seminoma occur within two years, as in non-seminoma. However few, late relapses after more than six years of surveillance have been reported and

the long-term relapse rate with adjuvant carboplatin is unknown. (9–12) Follow up is therefore recommended up to 10 years for these patients.

1.5 Aim

SWENOTECA VII is a cancer care program for seminomatous germ cell tumours in all stages in Sweden and Norway (part of). It is a continuation of the SWENOTECA V program and has mainly been revised with regard to treatment options in clinical stage I.

2 Aim of the SWENOTECA VII treatment program for patients with seminomatous testicular cancer

- To establish a complete register including all adult patients with seminomatous testicular cancer in Norway and Sweden.
- To standardise investigation procedures, staging and risk estimates, treatment and follow up.

2.1 Clinical stage I

- To compare the outcome of patients treated with one cycle of carboplatin with patients with no adjuvant treatment concerning acute and late toxicity, relapse rate and time to relapse and survival.
- To confirm published data on relapse rate for presumed low-risk patients on surveillance.
- To confirm published data on relapse rate after one course of carboplatin in presumed high-risk patients.

2.2 Advanced disease

- To evaluate treatment outcome, relapse rate and pattern of relapse and overall survival for patients treated with chemotherapy.
- To evaluate side effects after treatment for advanced disease.

3 Diagnosis, pre- and post-orchietomy examinations, clinical staging

3.1 Diagnosis of Testicular Cancer

Testicular cancer usually presents as a painless, unilateral intrascrotal mass and is in the majority of cases diagnosed by palpation. Fewer than 10% will present clinical symptoms mimicking epididymitis.

Ultrasound of both the testicles should be performed, and exploration should be performed in all cases when non-invasive investigations cannot exclude a tumour.

Transscrotal fine needle aspiration or biopsy from the tumour should not be performed.

3.2 Serum tumour markers in seminoma

In contrast to non-seminomatous germ cell cancer, seminoma patients often lack elevated tumour markers.

Human chorionic gonadotropin (HCG) is slightly/moderately elevated depending on tumour volume in 20–50% of the patients. (13) A HCG-value of more than 200 IU should raise the suspicion of non-seminomatous germ cell components and the specimen should be reexamined.

Alphafoetoprotein, AFP is by definition not consistent with a seminoma diagnosis. The detection of significantly elevated levels of AFP implies that the tumour specimen should be reexamined with respect to non-seminomatous elements. Even if these are not found, the tumour should be considered and treated as a non-seminoma! However, one should be aware that reparative and infectious/viral processes of the liver may induce a slight increase in AFP. Rarely patients constitutionally may have an AFP level slightly above the normal range. A modest and **stable** AFP level might thus be compatible with a seminoma diagnosis.

Lactate dehydrogenase (LDH) is a widely distributed enzyme of glycolytic metabolism and is released by many tissue types following cell damage. Total serum LDH levels are elevated in about 80% of patients with metastatic seminomatous testicular cancer. (14)

Placental alkaline phosphatase (PLAP) is elevated in 50% of the patients with seminoma but is only analysed in a few laboratories in Sweden. The use of this marker is optional. PLAP may be falsely elevated in smokers.

3.3 Fertility measures and hormonal analyses

Cryopreservation of sperm should preferably be offered before orchiectomy or otherwise before start of any therapy although the detrimental effect of adjuvant chemotherapy or limited para-aortic radiotherapy is probably much smaller than with para-aortic and ipsilateral iliac field radiotherapy. (15)

Sexual hormones (LH/FSH, testosterone and SHBG) should be analysed before and after orchiectomy and during follow up. The serum for the hormone analyses should preferentially be taken in the morning or at least before noon (due to their circadian variations). It is important to detect and treat hormonal insufficiency both with regard to short- and long-term morbidity of hypogonadism.

3.4 Before orchiectomy

- All patients should be offered pre-orchiectomy sperm count with cryopreservation
- Serum levels of AFP, HCG, LDH, (PLAP)
- Serum levels of LH, FSH, testosterone and SHBG
- Ultrasound examination of both testicles
- General physical examination

3.5 Inguinal exploration and orchiectomy

An incision similar to that performed in patients with inguinal hernia is done. The anterior wall of the inguinal canal is divided, and the vas and spermatic vessels are dissected free at the internal opening of the inguinal canal. In most cases there is no doubt of the diagnosis and the spermatic vessels and the vas are divided immediately. The testis and epididymis with their surrounding tunica vaginalis are pushed out of the scrotum and dissected free from the scrotal wall. If any doubt of the diagnosis, the spermatic cord is clamped before mobilization

of the scrotal content and the tunica vaginalis is opened for direct testicular inspection. Frozen sections may be unreliable and in most cases orchiectomy should be performed without delay. The vas and the spermatic vessels are ligated and divided separately close to the peritoneal fold.

The specimen is immediately sent for definitive histology. If possible, send the specimen fresh on ice to the pathology department, otherwise place it in formalin. The urologist should not incise the specimen.

3.6 Biopsy of the contralateral testis

Biopsy of the contralateral testis should be done because of the risk of cancer in situ (CIS), and this is best done at the time of the orchiectomy (see Addendum, chapter 24). The testis should be held firmly and a transscrotal incision is made laterally on the tunica vaginalis, long enough to see it clearly. After incising the parietal layer, make a small incision in the tunica albuginea to allow testicular tubules to bulge out. Snip off a tuft of tubules cleanly with fine sharp scissors. Place the biopsy at once into a specimen pot containing formalin. While performing the biopsy, careful handling and placement in fixative is important to prevent mechanical damage. If it is of importance to evaluate not only CIS but also spermatogenesis, the biopsy must be put in Bouin's solution. Close the incision in the tunica and skin separately with interrupted 4–0 absorbable sutures.

3.7 Pathological examination of the testis

Macroscopic features and sampling:

- Side, testis size, **tumour size** and the macroscopic features of the tumour, such as macroscopic involvement of epididymis, spermatic cord and tunica vaginalis.
- Sampling: 1 cm² section for every cm of maximal tumour diameter, including normal macroscopic parenchyma (if present), tunica albuginea and epididymis selection of suspected areas. At least one proximal and one distal section of spermatic cord, plus any suspected area.

Microscopic features and diagnosis:

- Histological type, and specification of individual components according to the 2004 WHO classification.
(Comment: Spermatocytic seminoma is a distinct tumour of uncertain histogenesis and occurs in elderly patients. The benign character and good prognosis of the spermatocytic variant of seminoma should be acknowledged. (16) This seminoma entity is not included in the current protocol.)
- Presence or absence of **rete testis invasion**.
- Presence or absence of tumoural vascular invasion, tunica albuginea, tunica vaginalis, epididymis or spermatic cord invasion.
- Presence or absence of intratubular germinal neoplasia in non-tumoural parenchyma.
- pT category according to TNM 2002
see addendum
- Immunohistochemical studies of seminomas are rarely indicated, except in those cases that are overgrown by lymphocytes, resembling lymphoma. PLAP is invariably positive.

3.8 Clinical staging

- Serum levels of AFP, β -HCG, LDH (PLAP optional)
- Chest X-ray
- CT of thorax, abdomen/pelvis should be performed shortly after orchiectomy. If there is clinically any indication of advanced metastatic disease it should be done before orchiectomy.

If there is evidence of metastatic disease, the patient should be referred immediately to a regional oncology department for further evaluation and treatment.

3.9 Diagnostic radiography

Chest x-ray and CT-scan of the chest and abdomen/pelvis are mandatory in the staging procedure of all testicular cancers. PET scan is a sensitive and specific method to detect seminoma in abdominal nodes but is not to be used as a routine staging procedure. (17–18) Other investigations according to symptoms. Isolated skeletal involvement does occur in seminoma, although rare.

4 Clinical staging, prognostic classification and risk grouping

- Clinical staging according to the RME staging system (**addendum 18**)
- Prognostic classification according to IGCCCG and MRC (**addendum 19**)
- Risk grouping in clinical stage I according to the presence of none, one or two of the risk factors: tumour size > 4cm, invasion of rete testis.

5 Patient information and treatment decision

The written information (**addendum 20, 21**) regarding the treatment options for patients in stage I must be given to the patient with adequate time for remaining questions. The patient should always be offered a new consultation within a short time. Furthermore, written information should be given of the registration in the SWENOTECA database, and of external (SWENOTECA) monitoring of the case records.

In Norway the written information must be signed by the patient.

In Sweden the patient's consent is noted in his case records.

The patient should be treated and followed according to the same principles even if he does not consent to be registered with full name in the database.

Immediately after the informed consent has been given, and treatment or surveillance is decided, the SWENOTECA "Registreringsblankett" should be sent to:

- Kontor for klinisk kreftforskning, Kreftavdelingen, Haukeland Universitetssykehus, 5021 Bergen (Norwegian patients)
- Onkologiskt centrum, Universitetssjukhuset i Lund, 221 85 Lund (Swedish patients)

6 Principles of treatment of Clinical Stage I Seminoma

6.1 Surveillance

More than 80% of the patients in CSI will not need any treatment and since the rescue rate with chemotherapy early on is almost 100%, surveillance has emerged as an attractive strategy. Risk factors for relapse were in 2002 identified by pooling data from the four largest surveillance studies comprising 638 patients from four centers: Royal Marsden Hospital, Danish Testicular Cancer Study Group, PMH and the Royal London Hospital. From this retrospective study of risk factors for relapse after surveillance, tumour size > 4 cm and rete testis invasion emerged as independent adverse prognostic risk factors. (23) The overall risk of relapse with a median follow up of 7 years was 20%, with a 12.2%, 15.9% and 31.5% risk for none, one or two risk factors respectively.

6.2 Carboplatin

A new adjuvant approach is chemotherapy with single agent carboplatin. (5, 24–26) Carboplatin monotherapy with one or two cycles has been shown to reduce the relapse rate effectively in non-randomised trials. In a single center study Steiner et al treated 108 patients with two cycles of carboplatin (400 mg/m²) and only 2 (1.85%) relapsed, both in the retroperitoneum within the first year after a median follow up of 5 years. (27) The treatment was well tolerated with mild thrombocytopenia and mild gastrointestinal side effects.

Recently results from the large randomised trial of Oliver et al in 2005 were reported. In 1477 patients with a median follow-up of 4 years, one cycle of carboplatin (AUC 7) showed non-inferiority compared with radiotherapy regarding relapse-free survival. (6) Acute side effects were more pronounced after carboplatin therapy. Patterns of relapse differed with more abdominal retroperitoneal relapses after carboplatin chemotherapy (74% of the relapses) than after radiotherapy (9% of the relapses). Long-term results are however still lacking.

Dosage schedule of carboplatin: **see addendum 22.**

6.3 Radiotherapy

Until now standard adjuvant treatment of CSI seminoma has been radiotherapy to the para-aortic lymph nodes and the ipsilateral iliac nodes, hockey stick field, or dog-leg field which also include the inguinal nodes, or surveillance with treatment only in case of relapse. Efforts have been made to minimize the radiation field and dose in order to decrease the risk for potential long time side effects. The MRC Trial TE 10 randomised patients to either 30 Gy in 15 fractions given to a para-aortic field or given as a classical dog-leg field. No difference in overall relapse rate was found, but more pelvic recurrences were seen in the group treated with para-aortic field. However, reduced haemathological-, gastrointestinal- and gonadal toxicity was found in this group. (19) The study excluded patients with pT4 tumours and previous inguinal or scrotal surgery. The MRC Trial TE 18 compared 30 Gy given in 15 fractions versus 20 Gy given in 10 fractions to the para-aortic field and found them equally effective. (20) The relapse rate after adjuvant radiotherapy is low, 1–3%. (21–22)

7 Treatment recommendations in Clinical Stage I

Based on the results of adjuvant treatment with one cycle of carboplatin compared with standard radiotherapy and the identified risk factors for relapse, the SWENOTECA propose a risk-adapted strategy for treatment of CSI seminoma. (6, 23)

- For low-risk patients with 0–1 risk factors surveillance is recommended. The patient may also choose to receive 1 course of carboplatin.
- For high-risk patients with 2 risk factors adjuvant treatment with one course of carboplatin is recommended. The patient may also choose surveillance.
- Adjuvant radiotherapy is recommended for those who by any means are not suitable for chemotherapy or surveillance. **See addendum 23**
 - ⇒ Radiotherapy to the para-aortic field with 20 Gy given in 10 fractions is recommended.
 - ⇒ Radiotherapy should be given to para-aortic and ipsilateral iliac nodes in patients not able to fulfil adequate follow-up
 - ⇒ In patients with T4 tumours or previous ipsilateral, inguinal or scrotal surgery, radiotherapy to para-aortic, ipsilateral iliac and inguinal nodes should be considered.

8 Principles of Treatment in Clinical Stage IIA Seminoma

Radiotherapy, background

In CSII A a major concern is to accurately define the disease as truly metastatic i.e. if nodes are involved or not when visualized. The progression of disease is slower than in non-seminomatous cancer and repeated CT scans does not easily give an answer and biopsy might be difficult in nodes less than 1.5 cm. PET scan could be used to confirm involvement and several studies indicate a high sensitivity and specificity in seminoma. (17)

With limited disease (<2 cm) in the abdominal nodes, radiotherapy given to a para-aortic and ipsilateral iliac field is standard treatment. According to data in the literature a target dose of 25–30 Gy results in excellent local control with a relapse free survival of 95%. (28–30)

The remaining testis receives 1–3% of the total radiation dose. Doses less than 0.5 Gy usually cause a transient oligospermia, while higher doses cause azoospermia. If the dose is less than 1.5 Gy, a recovery is seen within 2 years. (31) Shielding of the remaining testicle **should** be used.

Other concerns relate to data on long-term morbidity, such as increased risk for cardiovascular events and increased risk of second malignancies. (32–34) However, all of these reports refer to patients irradiated to larger target volumes and radiation doses.

9 Treatment recommendations in Clinical Stage IIA

The target volume includes the para-aortic and ipsilateral iliac lymph nodes to a target dose of 27 Gy with 1.8 Gy per fraction x 15. **See addendum 23.**

10 Treatment of advanced seminoma (CS IIB – CS IV)

Background

There are no randomized studies on patients with stage IIB disease comparing radiotherapy and chemotherapy. The reported relapse rate with radiotherapy varies between 9–24%. All available data are based on small series of patients. (28, 35–37) The relapses after radiotherapy are predominately located outside the retroperitoneum. (28, 35–37) Even if the risk factors for subclinical disseminated disease in stage IIB disease are unknown it is reasonable to believe that tumour volume is of importance. Both radiotherapy and chemotherapy are viable alternatives.

For higher stages of seminoma there is international consensus on treatment with multiple courses of cisplatin based combination chemotherapy. (7,8) As patients with advanced seminoma are infrequent there are no randomised studies comparing different kinds of cisplatin-based chemotherapy. However, these patients were often included in protocols for low risk testicular cancer irrespective of histology. Results from these studies have shown that 4 EP is as effective as other cisplatin regimens. (38) No studies support the use of single agent carboplatin chemotherapy in advanced disease. (39–40)

In the International Germ Cell Consensus Classification (**see addendum 19**), a prognostic factor-based classification system for metastatic germ cell cancers, metastatic seminoma is classified as good or intermediate prognosis. No seminoma patients are classified as poor risk. Adverse prognostic factors are non-pulmonary visceral metastases, especially liver or brain. Also, presence of supra clavicular nodes and raised LDH (>two times the upper limit of normal) added negative prognostic information. (13, 41)

Seminomatous tumors are often characterised by slow clinical regression rate following chemotherapy. Residual tumour mostly consists of fibrotic or necrotic tissue (7, 42–45) In post-chemotherapy seminoma, a positive PET is highly predictive for the presence of viable tumour especially when using a ≥ 3 cm cut-off. A negative PET scan is excellent for the exclusion of disease in lesions ≥ 3 cm. PET can contribute to the management of residual seminoma lesions, especially in terms of avoiding unnecessary additional treatment for patients with non-regressing lesions ≥ 3 cm. (17–18, 46)

11 Treatment recommendations in advanced disease

- Four courses of EP (etoposide, 100 mg/m² d 1–5 d + cisplatin 20 mg/m² d 1–5) is recommended for good risk patients.
- If contraindications to chemotherapy or small tumour volume, treatment with radiotherapy to para-aortic and ipsilateral iliac lymph nodes, 1.8 Gy x 20 to 36 Gy might be an option in stage IIB.
- For patients in the intermediate prognostic group according to the IGCCCG, (extra-lymphatic, extra-pulmonary metastasis) 4 courses of BEP chemotherapy is recommended. PEI chemotherapy may be an option in patients with risk factors for bleomycin toxicity such as age > 40, decreased kidney and pulmonary function.
- Dose reduction and treatment delay should be avoided.
- Evaluation of response should be performed after 2 courses of therapy.

Residual mass

- Post-chemotherapy surgery or consolidating radiotherapy without histological verification of seminoma is not indicated.
- It is not possible to give exact recommendations of how often patients with residual tumour should be checked. There are no solid data on factors predicting viable tumour after cisplatin-based chemotherapy.
- Patients with regressing or persisting radiological findings after primary chemotherapy should be monitored with an appropriate radiological method (CT, MRI, US) and serum tumour markers.
- Check ups should be more intense the first 6 months, i.e. every second-third month.
- A PET scan is recommended if the residual mass is ≥ 3 cm and not regressing in order to identify viable seminoma in a residual mass. It should not be performed earlier than 2 months after completion of therapy due to the risk of false positivity.
- If positive PET scan, histological verification should be performed before consolidating therapy is decided upon. If viable tumour is found either radiotherapy or surgical removal of the tumour may be discussed.
- In case of stable residual mass with a negative PET scan no further measure has to be taken.
- Post-chemotherapy radiotherapy in case of viable residual tumour should be given to limited fields and to a total dose of 36–40 Gy given with 2 Gy fractions.
- In case of progressive residual tumour, histological confirmation has to be performed before salvage therapy is started. Progressive disease after initial therapy is treated either with surgery, radiotherapy or second line chemotherapy based upon individual data.

Not Valid

As these situations are rare it is recommended to discuss within the SWENOTECA network how to handle these patients.

12 Follow-up principles

All seminoma patients are to be followed closely according to the follow-up schedule specified (**see addendum 25**). Follow-up during year 1–2 is at 4-monthly intervals, year 3–4 every six months and year 5–10 once a year except for patients treated with adjuvant radiotherapy who are followed for a total of 6 years. In patients treated with adjuvant radiotherapy radiological investigation of abdominal lymph node stations should be performed **only** at month 24. For those treated with para-aortic field only, **pelvic radiology** should be done **yearly**. In case of residual masses follow-up should be intensified. The follow-up schedule should be modified according to metastatic sites.

After completed follow-up (6 and 10 years respectively), a written patient information with regard to long-term effects should be given to the patient (go to www.ocsyd.lu.se).

It is generally desirable to reduce the radiation dose from repeated diagnostic imaging procedures to the patient without compromising the quality of follow-up. Ultrasound examination or magnetic resonance imaging (MRI) of the abdominal and pelvic lymph node areas may be performed when suitable. If there is any ambiguity, a computed tomography (CT) examination must be performed. CT or MRI is recommended at least once yearly. The

clinician has to choose the best technique available at the respective centre and when necessary, refer the patient to another more specialized centre. If MRI is used the principles of the imaging protocol in **addendum 26** must be followed. A dialogue with the responsible radiologist is necessary to make sure that the principles of the protocol and reasons for the follow-up is fully understood.

18F Fluorodeoxyglucose Positron Emission Tomography (FDG-PET) or PET/CT are still not established as routine imaging techniques in seminoma but may be used in special situations. PET can contribute to the management of residual seminoma lesions, especially in terms of avoiding unnecessary additional treatment for patients with non-regressing lesions ≥ 3 cm. For the evaluation of residual tumour a PET scan should not be performed earlier than 2 months after completion of therapy because of the risk of false positivity. (46)

Hormonal status is measured before and after orchiectomy and at 1, 3, 5 and 10 years or when needed.

The SWENOTECA VII seminoma follow-up form must be filled in and sent to the national SWENOTECA secretariat, preferably after each visit, or **at the least once a year** for the relapse free patient.

If some of the follow-up consultations are referred to a local hospital, the responsible SWENOTECA clinician must ensure that good compliance to follow-up can be continued, and that any relapse is reported promptly to the SWENOTECA secretariat.

13 Relapse

In case of doubt, morphological verification should be performed.

Treatment depends on site of relapse and previous treatment. There are few reports on second line-chemotherapy, but long-term remission in about 50% of the patients has been reported. (47–49)

The SWENOTECA VII Follow-up form must be filled in, and sent to the national SWENOTECA secretariat immediately if a relapse is detected.

It is very important to detect any deviation from the postulated relapse rates and patterns as early as possible, in order to adjust the treatment and/or follow-up program, if indicated.

13.1 Relapse after surveillance or adjuvant carboplatin chemotherapy

In case of abdominal lymph nodal relapse < 2 cm in the surveillance group, radiotherapy 1.8 Gy x 15 to 27 Gy to para-aortic and ipsilateral iliac nodes is the treatment of choice. Chemotherapy is also possible (EP x 4), but not first choice.

Other relapses should be treated with 4 courses of EP-chemotherapy. In very advanced disease (intermediate risk) BEP chemotherapy should be used.

If contraindications to chemotherapy or small tumour volume (2–5 cm) in abdominal lymph nodal relapse, treatment with radiotherapy to para-aortic and ipsilateral iliac nodes 1.8 Gy x 20 to a total dose of 36 Gy might be an option.

13.2 Relapse after adjuvant radiotherapy

Relapses should be treated with 4 courses of EP-chemotherapy. In very advanced disease (intermediate risk) BEP chemotherapy should be used.

13.3 Relapse or progression after standard combination chemotherapy

Chemotherapy, surgery or radiotherapy may be applicable.

Chemotherapy with PEI (cisplatinum 20 mg/m² d 1–5 + etoposide 100 mg/m² d 1–5 d + ifosfamide 1200 mg/m² d 1–5).

Chemotherapy with TIP (paclitaxel 250 mg/m², d 1 + ifosfamide 1500 mg/m² d 2–5 + cisplatinum 25 mg/m² d 2–5). (49)

Radiotherapy 1.8 Gy per fraction to a total dose of 36–40 Gy.

13.4 High-dose chemotherapy

At present no data exist on high dose chemotherapy with stem cell support in seminoma patients in the salvage situation. Therefore in these rare situations we recommend discussions within the SWENOTECA network. See the SWENOTECA protocol for NSGCT for further information on procedures for drug administration.

14 Monitoring and reporting the results of the program

The SWENOTECA VII registration form must be sent to the national SWENOTECA secretariat immediately after inclusion and the SWENOTECA VII treatment form for patients receiving treatment at first follow-up visit after treatment. The SWENOTECA VII follow-up form should preferably be sent in after each follow-up visit or at least once a year if there is no relapse.

In cases of relapse it is important that a follow-up form regarding the relapse is sent to the national SWENOTECA secretariat as soon as possible.

A treatment form must be forwarded to the secretariat after completion of the salvage therapy.

15 Ethical considerations

Seminoma is curable close to 100% in patients with CS I and IIA disease. Surveillance in clinical stage I seminoma will result in a relapse in 20% of the patients all of which are expected to be salvaged by chemotherapy or radiotherapy. The over treatment of > 85% of the patients and the potential long-term sequelae with radiotherapy makes surveillance a reasonable option. Short course chemotherapy with one or two courses of carboplatin has changed the scenario offering an adjuvant treatment which is convenient and as effective as radiotherapy, at least with a median follow up of 4 years, but without presumed or few side effects. It is important to inform the patient of the risk factors and the available choices.

A common treatment protocol, a common quality database, and carefully performed monitoring of an experienced data management secretariat are important features in securing optimal survival in seminoma patients. The patients are, however, free to abstain from such external registration and monitoring.

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Not Valid

17 TNM - Pathological (p) and Clinical classification

T - Primary Tumour

The extent of the primary tumour is classified after radical orchiectomy; see pT.

pT - Primary Tumour

- pTX Primary tumour cannot be assessed (if no radical orchiectomy has been performed, TX is used).
- pT0 No evidence of primary tumour (e.g. histologic scar in testis).
- pTis Intratubular germ cell neoplasia (carcinoma in situ).
- pT1 Tumour limited to testis and epididymis without vascular/lymphatic invasion; tumour may invade tunica albuginea but not tunica vaginalis.
- pT2 Tumour limited to testis and epididymis with vascular/lymphatic invasion, or tumour extending through tunica albuginea with involvement of tunica vaginalis.
- pT3 Tumour invades spermatic cord with or without vascular/lymphatic invasion.
- pT4 Tumour invades scrotum with or without vascular/lymphatic invasion.

N - Regional Lymph Nodes

- NX Regional lymph nodes cannot be assessed.
- N0 No regional lymph node metastasis.
- N1 Metastasis with a lymph node mass 2 cm or less in greatest dimension or multiple lymph nodes, none more than 2 cm in greatest dimension.
- N2 Metastasis with a lymph node mass more than 2 cm but not more than 5 cm in greatest dimension, or multiple lymph nodes, any one mass more than 2 cm but not more than 5 cm in greatest dimension.
- N3 Metastasis with a lymph node mass more than 5 cm in greatest dimension.

M - Distant Metastasis

- MX Distant metastasis cannot be assessed.
- M0 No distant metastasis.
- M1 Distant metastasis.
 - M1a Non-regional lymph node or pulmonary metastasis.
 - M1b Distant metastasis other than to non-regional lymph nodes and lungs.

18 Clinical staging: Modified after the Royal Marsden Hospital staging system

CS I No evidence of metastases

CS Mk+ β -HCG persistently elevated (not declining according to its half-time), but no metastatic disease demonstrated

CS II Metastatic disease restricted to abdominal nodes

A Maximal transverse diameter <2 cm
 B Maximal transverse diameter 2–5 cm
 C Maximal transverse diameter >5–10 cm
 D Maximal transverse diameter >10 cm

CS III Supradiaphragmatic node involvement

For abdominal lymph nodes:

0 No metastases
 A–D According to CS II

CS IV Extra-lymphatic metastases

Lung substage:

L1 ≤ 3 metastases, no metastases >2 cm
 L2 >3 – ≤ 20 metastases, no metastases >2 cm
 L3 ≤ 20 metastases, >2 cm
 L4 >20 metastases

For abdominal lymph nodes:

0 No metastases
 A–D According to CS II

H+ Liver metastases
 Br+ Brain metastases
 Bo+ Bone metastases

Not Valid

19 Prognostic classification

Medical Research Council

Small volume disease: CS Mk⁺, II_{A-B}, III_{0-A-B}, L1-2

Large volume disease: CS II_{C-D}, III_{C-D}, IV_{C-D}, L1-L2

Very large volume disease: CS IV_{0-D}, L3-L4; extralymphatic extrapulmonary metastases (bone, liver, brain)

International Germ Cell Collaborative Consensus Classification

A prognostic factor-based staging system for metastatic germ cell cancers from the International Germ Cell Collaborative Consensus Classification Group (1997).

Non-seminoma	Seminoma
Good-prognosis group 56% of non-seminomas All of the following criteria: Testis/retroperitoneal primary No non-pulmonary visceral metastases AFP <1,000 µg/L β-HCG <5,000 IU/L LDH <1.5 x n 5-year survival 92%	90% of seminomas All of the following criteria: Any primary site No non-pulmonary visceral metastases Normal AFP Any β-HCG Any LDH 5-year survival 96%
Intermediate-prognosis group 28% of non-seminomas All of the following criteria: Testis/retroperitoneal primary No non-pulmonary visceral metastases AFP >1,000 and <10,000 µg/L or β-HCG >5,000 and <50,000 IU/L or LDH >1.5 and <10 x n 5-year survival 80%	10% of seminomas Any of the following criteria: Any primary site Non-pulmonary visceral metastases Normal AFP Any β-HCG Any LDH 5-year survival 72%
Poor-prognosis group 16% of non-seminomas Any of the following criteria: Mediastinal primary Non-pulmonary visceral metastases AFP >10,000 µg/L or β-HCG >50,000 IU/L or LDH >10 x n 5-year survival 48%	No patients classified as poor prognosis

Notes: AFP = alpha-fetoprotein; β-HCG = beta-human chorionic gonadotropin; LDH = lactate dehydrogenase

20 Patientinformation

SWENOTECA 2007-01-30

Information till patienter med testikelcancer av typ seminom utan tecken till spridning, stadium I.

Du har nyligen blivit opererad för cancer i testikeln. Mikroskopisk undersökning av tumören har visat att det rör sig om typen seminom. Utredning med blodprover och röntgenundersökningar har inte påvisat någon spridning av tumören och Din tumör klassificeras då som stadium I.

Hos en del patienter med testikelcancer i stadium I, kan det trots detta föreligga icke påvisbar spridning av tumörceller, oftast till lymfkörtlarna längs ryggraden i buken (hos ca 15–20 %). Hos dessa 15-20% av patienterna kommer sjukdomen att återkomma vilket upptäcks när tumörcellerna i lymfkörtlarna tillväxer. Hos enstaka patienter kan också sjukdomen sprida sig till andra organ.

Vad det gäller den fortsatta behandlingen för din sjukdom finns enligt europeiska riktlinjer tre alternativ: enbart kontroller tills tecken på eventuellt återfall, en kur med förebyggande cytostatikabehandling (cellgift), eller strålbehandling.

Idag kan man inte på förhand veta vilka patienter som utan tilläggsbehandling kommer att förbli friska (80–85 %), eller vilka som kommer få återfall av sin sjukdom (15-20 %). Med tilläggsbehandling minskar man risken för återfall men samtidigt behandlas de 80–85 % som ändå inte skulle fått återfall, i onödan.

Vid ett återfall av testikelcancer finns effektiv behandling. Oavsett vilket alternativ du väljer är prognosen god.

Risicanpassade behandlingsalternativ

Man vet idag att riskfaktorer såsom tumörstorleken (>4cm) och tumörens växtsätt (inväxt i kanalsystemet i testikeln) ökar risken för återfall. Behandlingsalternativen styrs av följande:

0-1 riskfaktorer:

Om man inte har någon riskfaktor (tumörstorlek, växtsätt) eller bara en av dem så är återfallsrisken ca 15%, varför enbart kontroller är ett alternativ vi rekommenderar.

Du har dock möjlighet att välja att få tilläggsbehandling om du så önskar (en kur cytostatika eller strålbehandling)

2 riskfaktorer:

Om man har båda riskfaktorerna är risken för återfall i sjukdomen ca 30%. Vår rekommendation är att man behandlar med en kur cytostatika i förebyggande syfte. Risken för återfall minskar från 30% till ca 5%.

Du har dock möjlighet att välja enbart kontroller eller eventuellt strålbehandling.

Behandlingsalternativ: för- respektive nackdelar

Enbart kontroller

Fördelen är att ingen behandlas i onödan. Man ger ingen efterbehandling, utan Du blir i stället noga kontrollerad, var fjärde månad de två första åren och därefter med glesare mellanrum under cirka 10 år. Nackdelen är att om man får ett återfall så blir behandlingen för detta mer omfattande än vid förebyggande behandling. Ett litet återfall i lymfkörtlarna i buken behandlas oftast med strålbehandling i 15 dagar. Annan typ av återfall behandlas med 4 cytostatikakurer (se nedan).

En cytostatikakur

En kort förebyggande cytostatikabehandling med ett läkemedel (Karboplatin), samt därefter kontroller med blodprov och röntgenundersökningar var fjärde månad de två första åren och därefter med glesare mellanrum under cirka 10 år. Behandlingen ges polikliniskt under ca 30 minuter i ett dropp i armens blodkärl. Den mest besvärande biverkan är illamående som dock till största delen förhindras av mediciner mot illamåendet. Antalet vita blodkroppar sjunker de första veckorna efter behandlingen vilket medför att man kan bli infektiöskänslig. Även blodplättarna kan sjunka något, men sällan så att det får några konsekvenser för blodlevringen. Efter ca 3 veckor brukar man vara återställd. Behandlingen leder i allmänhet inte till håravfall.

Fördelarna med att ge en kur med förebyggande cytostatika är att de flesta återfall förhindras, samtidigt som risken för bestående biverkningar efter en kur cytostatika är liten.

Strålbehandling mot lymfkörtlarna längs ryggraden i buken.

Strålbehandlingen pågår i två veckor (totalt 10 behandlingar med 5 behandlingar per vecka). En av biverkningarna vid behandlingen är illamående men detta kan vanligtvis förhindras med modern behandling mot illamåendet. Andra biverkningar kan vara trötthet och diarré. Kontroller sker regelbundet under cirka 6 år.

Risken för återfall i testikelcancer efter sådan strålbehandling är mindre än 5 %.

Det finns en viss risk för att denna behandling på lång sikt kan leda till en ny cancerform hos enstaka individer. Hur stor denna risk är vet man inte säkert då modern strålbehandling är olik den som man gav tidigare. Tidigare gavs en högre stråldos. Man vet därför inte säkert om de undersökningar, som har påvisat ökad förekomst av ny cancer gäller för dem som får behandling i dag med en lägre stråldos. Detta behandlingsalternativ förordas därför inte i första hand.

Not Valid

Cytostatikabehandling vid återfall:

Den cytostatikakur man använder vid ett eventuellt återfall benämns EP-kur (eventuellt BEP-kur) och består av två, (respektive tre) olika cytostatika. EP-kur ges som ett intravenöst dropp av medicinerna cisplatin och etoposid i fem dagar (vid BEP-kurer ges dessutom bleomycin en gång i veckan). Man vårdas inne på sjukhuset 5 dagar vid varje behandlingstillfälle. Det vanligaste är att man ger fyra kurer med tre veckors mellanrum.

All cytostatikabehandling ger akuta biverkningar, som någon enstaka gång kan vara allvarliga. Den mest besvärande biverkningen är oftast illamående och kräkningar. Detta kan som regel förhindras och alltid begränsas genom modern illamåendebehandling. Oftast kommer ett håravfall två till fyra veckor efter den första cytostatikabehandlingen och efter 3 – 4 kurer kommer man att ha tappat allt hår. Håret växer ut igen några veckor efter att sista kuren givits. Antalet vita blodkroppar kommer att sjunka de första två veckorna efter behandlingen och man kan på grund av detta vara mer känslig för infektioner. Även andra blodvärden kan påverkas, men efter cirka tre veckor har de i allmänhet normaliserats. En del patienter känner en besvärande trötthet som kan vara bestående mellan kurer och cirka en månad efter sista kuren.

Det är också känt att 3–4 cytostatikakurer kan ge kvarstående biverkningar i form av hörsel och njurfunktionsskador samt påverkan på fertiliteten.

De olika alternativen har sina fördelar och nackdelar således. Vi önskar därför att Du noga läser igenom denna information och funderar över vad Du tror skulle passa Dig bäst. Vi lägger stor vikt vid eventuella önskingar från den enskilde patienten.

Vi vill be om ditt tillstånd att, inom de sekretesskyddade databaser vi har, få registrera uppgifter som rör din sjukdom, behandlingen och förloppet av sjukdomen. Vi kan också vid behov behöva gå igenom dina journalhandlingar och tumörpreparatet, för att komplettera dessa uppgifter som vanligen lämnas på förtryckta blanketter. Denna registrering har betydelse för att möjliggöra kontinuerlig utvärdering av olika behandlingsalternativ, samt för att framöver kunna dra slutsatser som skall leda till att bästa tänkbara behandling ges till patienter med testikelcancer.

Om du inte samtycker skall du meddela detta till oss.

.....
kontaktperson

.....
telefonnummer

Not Valid

21 Pasientinformasjon

SWENOTECA 2007-01-30

Informasjon til pasienter med testikkelkreft av typen seminom uten tegn til spredning, stadium I.

Du har nylig blitt operert for kreft i testikkelen. Undersøkelse av svulsten har vist at det dreier seg om typen seminom. Utredning med blodprøver og røntgenundersøkelser har ikke påvist noen spredning og svulsten din klassifiseres da som stadium I.

Hos noen pasienter med testikkelkreft i stadium I, kan det likevel foreligge mikroskopisk spredning av svulstceller, som oftest til lymfeknutene langs ryggraden i buken (hos ca. 15–20%). Hos disse vil antall svulstceller i lymfeknutene øke, og senere kunne oppdages ved røntgenundersøkelser. Hos enkelte pasienter kan sykdommen også spre seg til andre organ.

Når det gjelder den videre behandling av din sykdom (etter operasjon av testikkelen), finnes det ifølge europeiske retningslinjer tre alternativ: 1. kun kontroller, 2. forebyggende behandling med én kur cellegift eller 3. forebyggende behandling med strålebehandling.

I dag kan man ikke på forhånd vite hvilke pasienter som uten tilleggsbehandling kommer til å forbli friske (80–85%), eller hvem som kommer til å få tilbakefall av sin sykdom (15–20%). Med tilleggsbehandling reduserer man risikoen for tilbakefall, men dette innebærer også unødvendig behandling av de 80–85 % som ikke vil få tilbakefall.

Om det tilkommer tilbakefall av testikkelkreften, finnes det effektiv behandling. Det betyr at uansett hvilket alternativ du velger er prognosen god.

Risikotilpassede anbefalinger

Man vet i dag at risikofaktorer som størrelsen av svulsten (≥ 4 cm) i testikkelen og svulstens vekstmåte (innvekst i kanalsystemet i testikkelen) øker risikoen for tilbakefall. Anbefalt oppfølgings- evt. behandlingsstrategi styres etter følgende:

0-1 risikofaktorer:

Dersom man har ingen eller kun én risikofaktor (tumorstørrelse, vekstmåte), er tilbakefallsrisikoen ca. 15 %. I denne situasjonen vil vi anbefale kun kontroller.

Du har imidlertid mulighet til å velge å få tilleggsbehandling (én kur cellegift eller strålebehandling) dersom du ønsker dette.

2 risikofaktorer:

Dersom man har begge risikofaktorene er risikoen for tilbakefall av sykdommen ca. 30 %. I denne situasjonen er vår anbefaling én kur cytostatika i forebyggende hensikt. Risikoen for tilbakefall reduseres da fra 30 % til ca 5 %.

Du har imidlertid mulighet til å velge kun kontroller, eventuelt strålebehandling.

Behandlingsalternativer: fordeler og ulemper

Bare kontroller

Fordelen er at ingen behandles unødvendig. Man gir altså ingen etterbehandling, men du blir i stedet nøye kontrollert, hver fjerde måned de to første årene og deretter med lengre intervaller i cirka ti år. Ulempen er at dersom det kommer et tilbakefall, blir behandlingen for dette mer omfattende enn ved forebyggende behandling. Et lite tilbakefall i lymfeknutene i buken behandles likevel vanligvis med strålebehandling i 15 dager. Annen type tilbakefall behandles med 4 cellegiftkurer (se nedenfor).

En cellegiftkur

Dette innebærer behandling med én cellegiftkur (Karboplatin), fulgt av kontroller med blodprøver og røntgenundersøkelser hver fjerde måned de to første årene og deretter med lengre intervaller i cirka ti år. Behandlingen gis poliklinisk i løpet av ca. 30 minutter gjennom en vene på armen. Den mest besværende bivirkning er kvalme som i stor grad forebygges ved hjelp av kvalmestillende medisin. Antall hvite blodlegemer synker de første ukene etter behandlingen. Dette medfører at man kan bli mer utsatt for infeksjoner. Også blodplatene kan synke noe, men sjelden så mye at dette medfører risiko for blødninger. Etter 2-3 uker er vanligvis blodverdiene normalisert. Behandlingen gir vanligvis ikke håravfall. Fordelene med å gi en forebyggende cellegiftkur er at de fleste tilbakefall forhindres, samtidig som risikoen for senere bivirkninger etter en kur med cytostatika er liten.

Strålebehandling mot lymfeknutene langs ryggraden i buken.

Strålebehandlingen pågår i to uker (totalt 10 behandlinger/5 behandlinger pr uke). En av bivirkningene ved behandlingen er kvalme, men dette kan vanligvis forebygges med moderne kvalmebehandling. Andre bivirkninger kan være tretthet og diaré. Oppfølgende kontroller pågår i 6 år.

Risikoen for tilbakefall av testikkelkreft etter slik strålebehandling er mindre enn 5 %.

Det er en viss risiko for at denne behandling på lang sikt kan føre til en ny kreftform hos enkelte individer. Hvor stor denne risikoen er, vet man ikke sikkert fordi moderne strålebehandling er ulik den som ble gitt tidligere, da man ga høyere stråledoser. Man kan derfor ikke sikkert vite om de undersøkelser som har påvist økt forekomst av ny kreft, gjelder for dem som får behandling i dag med en lavere stråledose. Av denne grunn blir dette behandlingsalternativet ikke anbefalt som første valg.

Cellegiftbehandling ved tilbakefall

Den cytostatikakuren man bruker ved et eventuelt tilbakefall kalles EP-kur (eventuelt BEP-kur) og består av to (tre) ulike cytostatika. EP-kuren gis (gjennom blodet) i form av medikamentene cisplatin og etoposid i fem dager (ved BEP-kurer gir man dessuten bleomycin to ganger i uken). Man er innlagt i sykehus i 5 dager ved hver kur. Det vanligste er at man gir fire kurer med tre ukers mellomrom.

All cellegiftbehandling gir akutte bivirkninger, som enkelte ganger kan være alvorlige. Den mest ubehagelige bivirkningen er oftest kvalme og brekninger. Dette kan forhindres og alltid reduseres ved moderne kvalmebehandling. Oftest kommer det håravfall to til fire uker etter den første cytostatikabehandlingen. Etter 3–4 kurer vil man miste alt hår. Håret begynner å vokse ut igjen noen uker etter siste kur.

Antall hvite blodlegemer kommer til å synke de første to ukene etter behandlingen, og man kan på grunn av dette være mer utsatt for infeksjoner. Også andre blodverdier kan påvirkes, men etter ca. tre uker er disse vanligvis normalisert. En del pasienter kan føle en plagsom tretthet som kan vedvare mellom kurene og ca. en måned etter siste kur.

Det er også kjent at 3–4 cytostatikakurer kan gi langtidsbivirkninger i form av hørsel- og nyrefunksjonsskader, samt gi redusert sædkvalitet.

De ulike behandlingsalternativer har sine fordeler og ulemper. Vi ønsker derfor at du nøye leser gjennom denne informasjonen og vurderer hva du tror er det beste alternativet for deg. Vi legger stor vekt på eventuelle ønsker fra den enkelte pasient.

Not Valid

Konfidensialitet og dataregistrering

Forskningsmedarbeiderne har taushetsplikt på linje med de som behandler deg i sykehuset. Dataregisteret er sikret mot uvedkommende innsyn etter retningslinjer i norske helselover.

Behandlingsprotokollen og dette informasjonsskriv er godkjent av Regional komité for medisinsk forskningsetikk.

Vi ber om din tillatelse til å få registrere relevante opplysninger omkring din sykdom, behandlingen og det videre sykdomsforløp. Disse opplysningene lagres i den sikkerhetsbelagte medisinske databasen som er opprettet ved Kontor for Kliniske Kreftforskning, Haukeland Universitetssykehus, Bergen. Vi ber også om din tillatelse til at forskningsmedarbeidere herfra kan se gjennom din journal for å komplettere og kontrollere de registrerte opplysninger. Slik registrering og kontroll er nødvendig for kontinuerlig å vurdere fordeler og ulemper ved ulike behandlingsalternativer, og dra erfaringer som kan lede til best mulig behandling for pasienter med testikkelkreft. Etter vår mening vil en slik løpende registrering og mulighet for ekstern kontroll innebære ekstra kvalitetssikring også for ditt eget etterkontroll-opplegg.

Du kan reservere deg mot en slik person-identifiserbar registrering og ekstern kontroll av dine journalopplysninger. Vi ønsker i så fall å kunne registrere relevante opplysninger om deg i databasen kun via et løpenummer, slik at din identitet kun er kjent av din behandlende sykehusavdeling.

SAMTYKKERKLÆRING

Jeg gir tillatelse til at medisinske opplysninger som er relevante for min behandling og etterkontroll av min sykdom registreres med mitt navn og personnummer i en medisinsk database ved Kontor for Klinisk Kreftforskning (KKK) ved Kreftavdelingen, Haukeland Universitetssykehus, Bergen.

Ved behov kan forskningsmedarbeider fra KKK få kontrollere relevante opplysninger i min sykehusjournal.

Navn..... den...../..... 20.....
blokkbokstaver

.....
signatur

Lege..... den...../..... 20.....
blokkbokstaver

.....
signatur

Not Valid

Karboplatin

Seminom, adjuvant behandling

Preparat	Dos/ dostillfälle mg/m ²	Maxdos/ dostillfälle mg	Antal doser/ dygn	Dos interv. tim	Antal doser/ cykel	Administreringssätt	Dag
1. Karboplatin	7×(GFR+25)*		1		1	iv inf 30 min	1

*totaldos

Calverts formel: Dos = AUC x (GFR + 25)

AUC = 7 mg/ml × min

GFR = ml/min, okorrigerat värde

Dos = mg, totaldos

Prep

1 **1**

Ny cykel
↓

Dag **1** **22**

Cykellängd: 21 d

Notera!

Beredning och administrering v g v

Speciella åtgärder

Iohexolclearance för beräkning av GFR före behandlingsstart. S-kreatinin före varje cykel. Om s-kreatinin stiger >20 % göres iohexolclearance.

Dosreduktionsrekommendationer
Granulocyter $\times 10^9/L$ TPK $\times 10^9/L$

Vid cykelstart:

 <1.0 <100

Preparat, % av fulldos

1

Behandlingen oppskjutes

Anmärkning

Ingår i vårdprogram för seminom, SWENOTECA VII.

Karboplatin seminom

Blandning och administrering

Preparat	Blandas i ml	Administrering sätt	Administrering tid	Sköljdropp NaCl, ml	Spoldropp NaCl, ml	Kemiskt stabil	Kommentar
Karboplatin	250 5% glukos	iv inf	30 min	250			72 tim, kallt

Not Valid

EP

Testikelcancer, seminom

Preparat	Dos/ dostillfälle mg/m ²	Maxdos/ dostillfälle mg	Antal doser/ dygn	Dos interv. tim	Antal doser/ cykel	Administreringssätt	Dag
1. Etoposid	100		1		5	} iv inf 2 tim	1–5
2. Cisplatin	20		1		5		

Prep

11111

22222

Ny cykel

↓

Dag

12345

22

Cykellängd: 21 d

Beredning och administrering v g v

Speciella åtgärder

S-kreatinin inför varje cykelstart. Om patologiskt utföres iohexol-clearance.

Cisplatin gives med forcerad diures.

CAVE! aminoglykosid skall ej givas under eller en månad efter cisplatinbehandling.

Not Valid

Dosreduktionsrekommendationer

Granulocyter × 10⁹/LTPK × 10⁹/L

1.0–1.4 >50

< 1.0 <50

Clearance: ml/min 70–79

60–69

< 60

Preparat, % av fulldos

1

2

Ge behandling med G-CSF efter

Behandlingen uppskjutes, i första hand 3 dagar

100 100 dag 1–4

100 100 dag 1–3

100 ***

***cisplatin ersätts med karboplatin doserat enl Calverts formel:

Totaldos karboplatin, mg = 7 (okorrigerat clearance ml/min + 25)

Ex. okorrigerat clearance = 50 ml/min. 7(50 + 25) = 525 mg. Karboplatin gives dag 1.

Dock om nedsatt njurfunktion beror på tumörobstruktion gives fulldos cisplatin.

Anmärkning

Ingår i vårdprogram för seminomatös testikelcancer.

EP

Blandning och administrering

Preparat	Blandas i ml	Administrering sätt	tid	Sköljdropp NaCl, ml	Spoldropp NaCl, ml	Kemiskt stabil	Kommentar
				250			
Cisplatin	1000 NaCl	iv inf	2 tim			72 tim rumstemp.	
Etoposid							

Not Valid

Prehydrering:

1000 NaCl under 2 tim.

Hydrering under behandlingen:

500 ml Mannitol 15% gives under 1 tim parallellt med cisplatininfusion.

Under behandlingsdygnet gives ytterligare minst 2 000 ml vätska po el iv.

Posthydrering: dygnet efter sista cisplatininfusion minst 2 000 ml; om patienten ej själv kan dricka denna mängd, skall vätska givas iv.

Diuresen under behandlingsdygnet samt dygnet efter sista cisplatinbehandlingen skall vara > 400 ml/4 tim.

BEP**Testikelcancer, Icke epitelial ovarialcancer**

Preparat	Dos/ dostillfälle mg/m ²	Maxdos/ dostillfälle mg	Antal doser/ dygn	Dos interv. tim	Antal doser/ cykel	Administreringssätt	Dag
1. Bleomycin*	30 000 IE**		1		3	im/iv inj	1, 5, 16
2. Etoposid	100		1		5	iv inf 2 tim	1–5
3. Cisplatin	20		1		5		

* då patienten erhållit en kumulativ dos bleomycin på 300 000 IE gives regimen utan bleomycin

** totaldos

Prep

1

1

1

2

2

2

2

2

3

3

3

3

3

1

Ny cykel
↓

Dag

1

2

3

4

5

16

22

Cykellängd: 21 d

Beredning och administrering v g v

Speciella åtgärder

S-kreatinin inför varje cykelstart. Om patologiskt utföres ionexol-clearance.

Cisplatin gives med forcerad diures.

CAVE! aminoglykosid skall ej givas under eller en månad efter cisplatinbehandling.

Bleomycin: om toxisk reaktion vid bleomycintillförel (feber, frossa) gives steroider exempelvis Deltison 25 mg po eller 3–4 mg Betapred. Fortsättningsvis gives steroider profylaktiskt före bleomycin.

Dosreduktionsrekommendationer

Granulocyter $\times 10^9/L$ TPK $\times 10^9/L$

Preparat, % av fulldos

1 2 3

1.0–1.4	>50	Ge behandling med G-CSF efter		
< 1.0	<50	Behandlingen uppskjutes, i första hand 3 dagar		
Clearance: ml/min	70–79	100	100	100 dag 1–4
	60–69	50	100	100 dag 1–3
	<60	50	100	***

***cisplatin ersätts med karboplatin doserat enl Calverts formel:

Totaldos karboplatin, mg = 7 (okorrigerat clearance ml/min + 25)

Ex. okorrigerat clearance = 50 ml/min. $7(50 + 25) = 525$ mg. Karboplatin gives dag 1.

Dock om nedsatt njurfunktion beror på tumörobstruktion gives fulldos cisplatin.

Anmärkning

Ingår i vårdprogram för non-seminomatös testikelcancer.

Bleomycin: CAVE! Risk för allvarlig pneumonit föreligger. Var observant på tecken på pneumonit. Ökad risk vid ackumulerad totaldos >400 000 IE; hos äldre patienter; vid hög O₂-koncentration i inandningsluft; tidigare eller samtidig strålbehandling mot thorax; nedsatt njurfunktion.

BEP**Blandning och administrering**

Preparat	Blandas i ml	Administrering sätt	Administrering tid	Sköljdropp NaCl, ml	Spoldropp NaCl, ml	Kemiskt stabil	Kommentar
Cisplatin	1000 NaCl	iv inf	2 tim	250			72 tim rumstemp.
Etoposid							
Bleomycin							
		im					7 dygn, kallt

Not Valid

Prehydrering:

1 000 NaCl under 2 tim.

Hydrering under behandlingen:

500 ml Mannitol 15% gives under 1 tim parallellt med cisplatininfusion.

Under behandlingsdygnet gives ytterligare minst 2 000 ml vätska po el iv.

Posthydrering:

dygnet efter sista cisplatininfusion minst 2 000 ml; om patienten ej själv kan dricka denna mängd, skall vätska givas iv.

Diuresen under behandlingsdygnet samt dygnet efter sista cisplatinbehandlingen skall vara > 400 ml/4 tim.

PEI

Testikelcancer

Preparat	Dos/ dostillfälle mg/m ²	Maxdos/ dostillfälle mg	Antal doser/ dygn	Dos interv. tim	Antal doser/ cykel	Administreringssätt	Dag
1. Cisplatin	20		1		5	iv inf 2 tim	1–5
2. Etoposid	100		1		5		
3. Ifosfamid	1200		1		5		
4. Mesna	240 (20% av ifosf dos)		1		5	iv inf 30 min	1–5
Mesna	480 (40% av ifosf dos)		2		10		
						po*	2 o 6 tim efter ifosfamid

*Om patienten inte *säkert* får i sig mesna po (kräks) gives samtliga 3 doser iv. 20% av ifosfamiddosen gives då timme 4 och 8.

Prep

1

1

1

1

1

2

2

2

2

2

3

3

3

3

3

4

4

4

4

4

Ny cykel

↓

Dag

1

2

3

4

5

22

Cykellängd: 21 d

beredning och administrering v g v

Beredning och administrering v g v

Speciella åtgärder

S-kreatinin inför varje cykelstart. Om patologiskt utföres ionexol-clearance.

Cisplatin gives med forcerad diures.

CAVE! aminoglykosid skall ej givas under eller en månad efter cisplatinbehandling.

Dosreduktionsrekommendationer

Granulocyter × 10⁹/L TPK × 10⁹/L

1.0–1.4

>50

< 1.0

<50

Clearance: ml/min

60–69

< 60

Preparat, % av fulldos

1

2

3

4

Ge behandling med G-CSF efter

Behandlingen uppskjutes, i först hand 3 dagar

100 dag 1–3

100

100 dag 1–4

100 dag 1–4

**

100

100 dag 1–4

100 dag 1–4

**cisplatin ersätts med karboplatin doserat enl Calverts formel:

Totaldos karboplatin, mg = 7 (okorrigerat clearance ml/min + 25)

Ex. okorrigerat clearance = 50 ml/min. 7(50 + 25) = 525 mg. Karboplatin gives dag 1.

Dock om nedsatt njurfunktion beror på tumörobstruktion gives fulldos cisplatin.

Anmärkning

Ingår i vårdprogram för non-seminomatös testikelcancer.

PEI

Blandning och administrering

Preparat	Blandas i ml	Administrering sätt	tid	Sköljdropp NaCl, ml	Spoldropp NaCl, ml	Kemiskt stabil	Kommentar
Cisplatin	1000 NaCl	iv inf	2 tim	250			72 tim rumstemp.
Etoposid							
Ifosfamid	250 NaCl	iv inf	30 min				72 tim, kallt
Mesna 1:a dos							

Mesna dos 2 och 3 gives om möjligt po

Not Valid

Prehydrering:

1 000 NaCl under 2 tim.

Hydrering under behandlingen:

500 ml Mannitol 15% gives under 1 tim parallellt med cisplatininfusion.

Under behandlingsdygnet gives ytterligare minst 2 000 ml vätska po el iv.

Posthydrering: dygnet efter sista cisplatininfusion minst 2 000 ml; om patienten ej själv kan dricka denna mängd, skall vätska givas iv.

Diuresen under behandlingsdygnet samt dygnet efter sista cisplatinbehandlingen skall vara > 400 ml/4 tim.

TIP – Testikelcancer

Blandning och administrering

Preparat	Blandas i ml	Administrering sätt	Administrering tid	Sköljdropp NaCl, ml	Spoldropp NaCl, ml	Kemiskt stabil	Kommentar
Paclitaxel	1000 NaCl	iv inf	24 tim	250			48 tim, rumstemp
Ifosfamid	1000 NaCl	iv inf	2 tim	250			72 tim, kallt
Mesna 1:a dos							
Mesna följande doser		iv inj/po					
Cisplatin	500 NaCl	iv inf	1 tim				72 tim, rumstemp

Not Valid

Hydrering under behandlingsdygn 2–5:

500 ml 15% Mannitol ges parallellt med cisplatin inf under 1 tim.

Posthydrering:

2000 ml NaCl ges efter cisplatin infusionen

Diures under behandlingsdygn 2–5 skall vara >400 ml/4 tim.

23 Radiotherapy

Dose:

CSI: 2 Gy x 10, 5 days weekly to total dose of 20 Gy to the para-aortic lymph nodes.

Note: If T4 tumour or previous inguinal or scrotal surgery, treatment of the para-aortic, the ipsilateral common and external iliac, and the ipsilateral inguinal lymph nodes should also be considered.

CSII A: 1.8 Gy x15, 5 days weekly to total dose of 27 Gy to the para-aortic and the ipsilateral common iliac- and the external iliac lymph nodes

Patient position and fixation:

The patient is placed in the supine position, fixated according to local practice for reproducible positioning of the patient during the whole treatment process. Mark the orchiectomi scar with a pewter thread. For patients in reproductive age a lead shield should be used to protect the contra lateral testis from external scattered radiation.

Radiotherapy treatment technique:

A CT-based 3-dimensional (3D) planned radiotherapy is mandatory. The CT based plan of the fields is generated based on vascular anatomy as the lymph nodes follow the vessels (aorta, vena cava inferior, ipsilateral renal vein, the common iliac and external iliac vessels).

Beam quality:

Minimum 6 MV photons can be used.

Target volumes and organs-at-risk (OAR) volumes:

Target volumes

GTV (Gross tumour volume) should be defined as the volume of any lymph node enlarged due to metastasis (i.e., CSIIA).

CTV (Clinical target volume) in the para-aortic region should include the para-aortic lymph nodes from the upper border of the 11th thoracic vertebra to the aortic division and is defined as the combined inferior vena cava and aorta volume including visible lymph nodes and any GTV with an additional margin of 1.4 cm in the anterior, lateral, inferior and superior directions. Similarly for the renal vein volume except for no expansion laterally.

If the ipsilateral common iliac- and the external iliac lymph nodes are to be treated (i.e. CSIIA), the CTV should be extended to include the combined volume of the common iliac and external iliac vessels to the level of the top of the obturator foramen, including visible lymph nodes and any GTV, with an additional margin of 1.4 cm in all directions.

In case of previous inguinal or scrotal surgery or a T4 tumour, inclusion of both the ipsilateral common iliac- and the external iliac lymph nodes and the ipsilateral inguinal lymph nodes with additional margins as described in the former passage should be considered.

ITV (internal target volume) should be identical to the CTV as organ movement can be neglected.

PTV (planning target volume) is defined according to the ICRU definition.

Organs at risk:

The volumes of both kidneys should be outlined in each CT image. No more than 25% of each kidney volume should receive more than 20 Gy.

24 Carcinoma-in-situ of the testis

Definition

Testicular carcinoma-in-situ (CIS) represents a specific histological pattern characterised by presence in the seminiferous tubules of cells possessing several cellular characteristics of malignancy: abundant, glycogen-rich cytoplasm, large irregular nucleus with coarse chromatin clumps, aneuploid DNA content and multiple nucleoli (Skakkebak 1978). In typical cases, the CIS cells and normally looking Sertoli cells are the only cell types present in the tubules. However, in some cases the CIS cells can be spread among cells of normal spermatogenesis. The proportion of tubules containing CIS cells vary from few to near 100% and usually the CIS tubules are dispersed throughout the whole testis (Jacobsen GK. *et al.*, 1981).

Natural course

CIS was originally reported in two infertile men who subsequently developed testicular germ cell cancer (TGCC) (Skakkebak 1972). Studies of infertile men as well as TGCC patients harbouring CIS in the contralateral testis indicated that following the diagnosis, 50% of the men develop invasive tumours in 5 years and 70% in seven years (Giwerzman *et al.*, 1987). Spontaneous regression of CIS has not been reported, and it seems probable that the majority – if not all – cases of CIS progress into an invasive stage of TGCC (seminoma or non-seminoma).

Biological aspects

Epidemiological, immunohistochemical studies indicate that CIS has its origin in the early foetal life (Jørgensen *et al.*, 1993; Møller and Skakkebak 1993; Møller 1993). It has been suggested that CIS cells are foetal gonocytes which have undergone malignant transformation (Skakkebak *et al.*, 1987). The aetiology behind arise of CIS and thereby TGCC is unknown. The rapid increase in the incidence of TGCC, which has taken place during the past few decades, indicates an impact of environmental and/or life style related factors affecting the individual already in early foetal life (Skakkebak *et al.*, 2001). Mother smoking during the pregnancy and chemicals acting as ‘endocrine disrupters’ have been linked to the aetiology of the CIS. The fact that CIS probably arise in the early foetal life does also have practical clinical implications. Provided that the screening method has sufficient sensitivity (see below), if CIS is not found in an adult man, his subsequent risk of developing TGCC is negligible.

High risk groups

Danish studies have shown presence of CIS in 5–6% of men diagnosed with unilateral TGCC (von der Maase *et al.*, 1986). However, the high risk of CIS in the contralateral testis is not a Danish phenomenon only, since similar figure was also found in a German study (Dieckmann and Loy 1996). Unfortunately, corresponding figures from other countries are lacking.

Other high risk groups for CIS of the testis are: men with a history of cryptorchidism (2–3%) (Giwerzman 1992); patients with infertility problem due to impaired testicular function (3% in men with non-obstructive azoospermia); men with an extragonadal germ cell tumours (up to 50%) (Daugaard *et al.*, 1987); individuals with intersex conditions and gonadal dysgenesis (rare conditions with very high risk of gonadal malignancy) (Müller 1987).

Clinical features

CIS of the testis does not present with any specific clinical features. Testes harbouring CIS may be atrophic but CIS can also be found in normal-size gonads. Testicular palpation can be associated with some tenderness but usually is not associated with any abnormal findings. There are no specific serum markers of CIS.

Testicular ultrasound

Recent data have indicated that *testicular microlithiasis (TM)* - detection of multiple, small hyperechogenic lesions in the testis – is associated with high risk of CIS (Lenz *et al.*, 1987; von Eckardstein *et al.*, 2001). Thus, in a study of 78 men with TGCC the predictive value of TM for the contralateral testis to contain CIS was 22.2% (Lenz *et al.*, 1996). Although a single case of CIS was found in a testis without TM, in such case the predictive value that the testis would not contain CIS was 97.6% (Lenz *et al.*, 1996). This result was confirmed in another study (von Eckardstein *et al.*, 2001).

Recommendations for use of ultrasound and its implications:

- 1) *TGCC patients*: since testicular biopsy can easily be performed in these men at the time of orchidectomy, ultrasound as a screening for CIS in the contralateral testis is not recommended. However, in rare cases where the biopsy performed at the time of orchidectomy fails ultrasound can be used for decision whether to repeat the biopsy (+TM) or not (- TM);
- 2) In men seeking for infertility and presenting with testicular atrophy (one or both testes with orchidometer measure below 15 ml) and/or history of cryptorchidism, testicular biopsy is indicated if TM is found;
- 3) The clinical implications of finding TM in otherwise healthy men are yet unknown and the method is therefore not recommended as a general screening for early TGCC.

Not Valid

Diagnosis

The only reliable method of diagnosing CIS is performing open surgical biopsy (Dieckmann *et al.*, 1999). It has to be stressed that the sensitivity of a needle biopsy has not been investigated. In TGCC patients the biopsy can ideally be done at the time of orchidectomy. The risk of serious complications following open surgical biopsy is very low and significantly less than the probability of developing contralateral TGCC (Bruun *et al.*, 1987). **The advantage of diagnosing the malignancy at a pre-invasive stage is the possibility of preserving the testis and thereby the endogenous androgen production (Giwerzman *et al.*, 1991) (see below).** It is recommended that the biopsy is performed in all men undergoing orchidectomy, since no clinical or laboratory test are sufficiently reliable to exclude TGCC patients not being at risk of having CIS.

A single biopsy, approximately 3 mm in diameter disclose CIS in 95% of the cases when this condition is present (Dieckmann *et al.*, 1999). Therefore, provided that the risk of contralateral CIS is 5%, if no CIS is found the risk of subsequent second testicular tumour is less than 0.3%. However, such high sensitivity in the diagnosis of CIS can only be obtained if: a) the tissue is properly handled; b) the diagnosis is performed by a pathologists experienced in this area. A double-biopsy procedure may yield an even higher disclosure rate up to 99% (Dieckmann *et al.*, 2006, European Urology in press).

Place the biopsy at once into a specimen pot containing formalin. While performing the biopsy, careful handling and placement in fixative is important to prevent mechanical damage.

If it is of importance to evaluate not only CIS but also spermatogenesis, the biopsy **must** be put in Bouin's solution.

For pathological investigation, routine haematoxylin-eosin staining is sufficient. However, immunohistochemical staining with use of an antibody against Placental-like Alkaline Phosphatase can facilitate the diagnosis. It is important that the whole tissue block is thoroughly investigated since such procedure minimise the risk of missing the diagnosis of focal CIS.

Management of CIS

1) Unilateral TGCC and CIS in the contralateral testis:

- a) **In patients not receiving chemotherapy:** CIS cells can be eradicated by local irradiation given as 8 daily doses of 2 Gy (total dose – 16 Gy). Although some of the men subsequently develop androgen insufficiency, in more than 50% androgen replacement is not required, at least during the first years post-irradiation. **For details of radiation technique see page 47.**
- b) **In patients receiving chemotherapy:** Platinum containing chemotherapy may eradicate CIS. However, patients with CIS may develop invasive cancer in spite of chemotherapy (Christensen et al., 1998). The safest alternative is to give local irradiation as indicated under (a). However, an alternative is to repeat the biopsy, 1–2 years after completion of chemotherapy, and perform ultrasound every 6 months until biopsy. If CIS cells are present, irradiation should be offered. However, it should be kept in mind that following chemotherapeutic treatment the CIS cells may be reduced in number without being completely eradicated. A double biopsy is therefore recommended as the sensitivity of a single testicular biopsy is expected to be lower than the figures given above and the risk of late contralateral TGCC exists. Even if the rebiopsy is negative, testicular ultrasound should be performed once yearly during the follow-up;

2) *Patients with extragonadal disease and CIS in one testicle:* Orchidectomy of the affected testicle is recommended;

3) *Bilateral CIS:* Irradiation as indicated under 1 a;

4) *Unilateral CIS and no malignancy in the other testis:* Orchidectomy.

Guidelines for follow-up after testicular irradiation for CIS:

- Control testicular biopsy should be done 12–24 months after irradiation and should disclose Sertoli-cell only pattern. Presence of germ cells indicate failure of the radiotherapy;
- Serum levels of testosterone, SHBG, LH, FSH and oestradiol should be checked prior to the radiation therapy, 6 and 12 months after. Subsequently the tests should be repeated with 12–24 months interval. Symptoms of hypogonadism combined with subnormal or low normal (even men with testosterone levels within the reference interval can be hypogonadal) total and free (adjusted for SHBG level) testosterone as well as high LH can be indicative of need for androgen substitution;
- Testicular ultrasound should be performed 3 months after radiotherapy, and every second year during 10 years follow-up.

Treatment of CIS and preservation of fertility potential

Testicular irradiation will lead to eradication of all germ cells and permanent sterility.

Therefore, if the patient has a wish of future fertility following precautions should be taken:

- If the patient has a partner, immediate wish of having a child and significant sperm output (this issue needs to be discussed with an andrologist) some months to few years of surveillance, while the couple is trying to obtain pregnancy can be recommended. During this period testicular palpation and ultrasound should be performed every 6th month;
- In other cases cryopreservation of sperms prior to irradiation is recommended;
- In case of azoospermia (no sperms in the ejaculate) and a strong wish of preservation of fertility, multiple testicular biopsies, and if intratesticular elongated spermatids are found, subsequent cryopreservation is an option to be discussed with the patient.

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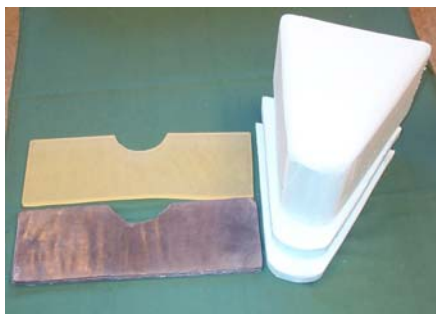
Not Valid

Behandling av kvarvarande testikel med elektroner

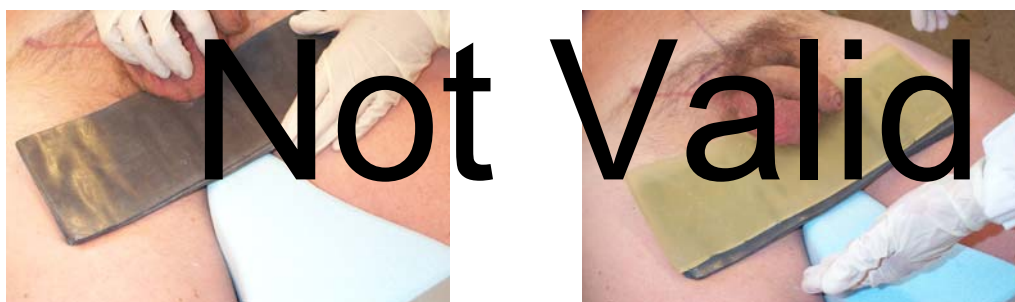
STRÅLKVALITÉ

Elektronenergi väljs så att **minimidosen** i testis är 16 Gy. Dos per fraktion: 2 Gy.

Uppläggning



För uppläggningsen behövs ett stöd för blyskyddet (t.ex. en trekant av frigolit), 1 cm tjockt vattenekvivalent bolus, 0.8–1 cm tjockt blyskydd samt ytterliggare bolus (ej med på bilden). Blyskyddet och det bolus som skall placeras under testikeln har en halvmåneformad urfasning för bättre passform mot scrotum.



Patienten placeras i ryggläge med benen fixerade brett isär.

Placera blystödet (trekanten av frigolit) mellan benen. Närmast under testikeln placeras 1 cm vattenekvivalent bolus följt av ett 0.8–1 cm tjockt blyskydd. Blyskyddet kan med fördel bestå av flera tunnare skivor.



Tejpa undan penis uppåt. Kring testikeln placeras vattenekvivalent bolus med en utsträckning på minst 2 cm lateralt. Det är viktigt att bolusen har en så bra passform som möjligt. Vid användandet av ”låga” elektronenergier kan det vara aktuellt att placera bolus även ventralt om testikeln.

25 Follow-up all seminoma patients

Schedule to be attached to patient chart, put date at checkpoint

Name: _____

ID-number: _____

Invasion rete testis: ☐ yes ☐ no Tumour size: _____

☐ Surveillance ☐ Carboplatin ☐ RT-paraaortic ☐ RT-paraaortic+ilical ☐ EP ☐ BEP ☐ Date end of treatment: _____

Type A: clinical invest, HCG, LDH, creatinin, AFP (optional), chest X-ray, abdominal and pelvic CT/MRI/US (see page 15 and 49)
(CT or MRI at least once yearly)

Type B: Like A plus, testosteron, SHBG, LH, FSH. Spermcount (optional)

NOTE!

Should be modified with regard to metastatic sites and residual tumour! Residual tumour after end of treatment should be followed with shorter intervals during the first 6 months (see page 15). PLAP is an optional tumour marker.

Patients in stage I treated with- paraaortic RT: pelvic CT/MRI is recommended yearly for six years, abdominal CT only month 24
- paraaortic + iliacal RT: abdominal and pelvic CT is needed only month 24

0 = date end of treatment		A	B	1st year
		8	12	Months after treatment
12	6	A	A	2nd year
		20	24	
24		A	B	3rd year
		30	36	
36		A	A	4th year
		42	48	
48			B	5th year
			60	
60			A	6th year
			72	

Patients in stage I treated with RT are followed for 6 years. Patients on surveillance or treated with adjuvant carboplatin, and patients treated for metastatic disease are followed according to type A year 7-10. Other investigations on indication.

After completed follow-up (6 and 10 years respectively), a written patient information with regard to long-term effects should be given to the patient (go to www.ocsyd.lu.se).

SWENOTECA VII, Seminom Registreringsblankett		Personnummer
Svenska patienter SWENOTECA sekretariatet Onkologiskt centrum Universitetssjukhuset i Lund 221 85 LUND Klinik, sjukhus Läkare		Namn
Norska patienter Kontor for klinisk kreftforskning Kreftavdelningen Haukeland Universitetssykehus 5021 Bergen		

Orchiektomi

☐ Nej ☐ Ja Datum år mån dag	Kontralateral testisbiopsi ☐ Cis ☐ ej Cis ☐ ej utfört	Spermigram utfört ☐ nej ☐ ja, om ja: ☐ före orchiektomi ☐ efter orchiektomi
Sjukhus	Patolog avd	PAD nr - år
Orchiektomi ☐ hö ☐ vä Inväxt i rete testis ☐ nej ☐ ja Vaskulär invasion ☐ nej ☐ ja ☐ oklar	Tumörstorlek x mm "Utbränd tumör" ☐ nej ☐ ja	Ifylles endast om tumörstorleken inte är angiven

Tumörmarkörer

Före orchiektomi Datum år mån dag	AFP , ☐ normalt ☐ förhöjt ☐ ej utfört β-HCG , ☐ normalt ☐ förhöjt ☐ ej utfört LD , ☐ normalt ☐ förhöjt ☐ ej utfört PLAP , ☐ normalt ☐ förhöjt ☐ ej utfört
Efter orchiektomi (vid definitiv stadiindelning) Datum år mån dag	AFP , ☐ normalt ☐ förhöjt ☐ ej utfört β-HCG , ☐ normalt ☐ förhöjt ☐ ej utfört LD , ☐ normalt ☐ förhöjt ☐ ej utfört PLAP , ☐ normalt ☐ förhöjt ☐ ej utfört
Hormonstatus Datum år mån dag	Testosteron , ☐ normalt ☐ förhöjt ☐ lågt ☐ ej utfört SHBG ☐ normalt ☐ förhöjt ☐ lågt ☐ ej utfört LH ☐ normalt ☐ förhöjt ☐ lågt ☐ ej utfört FSH ☐ normalt ☐ förhöjt ☐ lågt ☐ ej utfört

Metastaser

Lymfkörtelmetastaser Inguinalt ☐ nej ☐ ja Iliakalt ☐ nej ☐ ja Paraaortalt ☐ nej ☐ ja Mediastinalt ☐ nej ☐ ja Supraklav ☐ nej ☐ ja	Största metastas (mm x mm) x x x x x	Extralymfatiska metastaser Lunga ☐ nej ☐ ja Antal lungmetastaser Skelett ☐ nej ☐ ja Lever ☐ nej ☐ ja Hjärna ☐ nej ☐ ja Annat ☐ nej ☐ ja Lokal:	Största metastas (mm x mm) x x
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Klinisk stadiindelning¹ ☐ CSI Antal riskfaktorer ² T-stadium pT4 ³ ☐ 0 ☐ 1 ☐ 2 ☐ nej ☐ ja				☐ CSII ☐ CSIII ☐ CSIV ☐ CSMk+			
Abdominella lymfkörtlar ☐ 0 ☐ A ☐ B ☐ C ☐ D		Gruppering enl MRC⁴ ☐ small ☐ large ☐ very large volume		Prognos enl IGCC⁵ ☐ god ☐ intermediär ☐ dålig			
Behandling CSI ☐ surveillance ☐ adj. Karboplatin ☐ adj. strålbehandling							
Behandling CSII-IV ☐ strålbehandling ☐ EP ☐ BEP Annan, spec							

Förklaringar till Registreringsblankett SWENOTECA VII, Seminom

1. Klinisk stadiindelning: modifierad efter RMH

CS I Inga tecken på metastaser

CS Mk+ β -HCG kvarstående förhöjdt (faller ej enl sin halveringstid)
men inga metastaser påvisbara

CS II Metastaser begränsade till abdominella lymfkörtlar

A maximal diameter <2 cm

B maximal diameter 2–5 cm

C maximal diameter >5–10 cm

D maximal diameter >10 cm

CS III Metastaser i lymfkörtlar ovan diafragma

CS IV Extralymfatiska metastaser

För lungmetastaser gäller:

L₁ ≤ 3 metastaser, ingen >2cm

L₂ >3 – ≤ 20 metastaser, ingen >2cm

L₃ ≤ 20 metastaser, >2cm

L₄ >20 metastaser

För abdominella lymfkörtlar gäller:

0 inga metastaser

A–D enl ovan CS II

2. Riskfaktorer

Primärtumör >4 cm, inväxt i rete testis

3. pT4

Tumour invades scrotum with or without vascular/lymphatic invasion

4. Gruppering enl Medical Research Council

Small volume disease CS Mk+, II_{A–B}, III_{A–B}, IV_{0–A–B}, L1–L2

Large volume disease CS II_{C–D}, III_{C–D}, IV_{C–D}, L1–L2

Very large volume disease CS IV_{0–D}, L3–L4; extralymfatiska extrapulmonella metastaser
(skelett, lever, hjärna)

5. International Germ Cell Consensus Classification

Good-prognosis group. All of the following criteria:

Any primary site

No non-pulmonary visceral metastases

Normal AFP

Any β -HCG

Any LDH

Intermediate-prognosis group. Any of the following criteria:

Any primary site

Non-pulmonary visceral metastases

Normal AFP

Any β -HCG

Any LDH

Poor-prognosis group

No patients classified as poor prognosis

Not Valid

SWENOTECA VII, Seminom		Personnummer
Behandlingsblankett		Namn
Svenska patienter SWENOTECA sekretariatet Onkologiskt centrum Universitetssjukhuset i Lund 221 85 LUND	Norska patienter Kontor för klinisk kreftforskning Kreftavdelningen Haukeland Universitetssykehus 5021 Bergen	
Klinik, sjukhus		
Läkare		

Orsak till behandling		
☐ adjuvant	☐ metastatisk sjukdom	☐ recidiv
Start av behandling		Avslutad behandling
År mån dag		år mån dag
Kemoterapi ☐ Karboplatin ☐ EP ☐ BEP ☐ annat Antal kurer		
Strålbehandling ☐ paraaortalt ☐ paraaortalt + iliakalt ☐ annat Fraktionsdos , Gy Targetdos , Gy		
Orsak till avslutad behandling		
☐ enligt program ☐ terapivikt ☐ biverkningar ☐ annan, spec.....		
Toxicitet¹ Grad 3–4		
Hematol.	Hb ☐ nej ☐ ja	Perifer neuropati ☐ nej ☐ ja
	Vita ☐ nej ☐ ja	Obstipation ☐ nej ☐ ja
	Trombocyter ☐ nej ☐ ja	Infektion ☐ nej ☐ ja
Renal	S-kreat ☐ nej ☐ ja	Annan allvarlig toxicitet ☐ nej ☐ ja, spec
Effekt av behandling²		
☐ adjuvant beh ☐ ej utvärderat ☐ PR ☐ S ☐ PD ☐ ej utvärderat		
Fortsatt behandling (när anställd eller ruder ifylles när beslut fattats och eventuellt tilläggshandling givits)		
☐ ingen ☐ konsoliderande strålbehandling ☐ högdoskemoterapi med rescue ☐ kemoterapi, spec ☐ annan behandling, spec		
Konsoliderande strålbehandling (efter kemoterapi/kirurgi)		
Startdatum år mån dag Fraktionsdos , Gy Targetdos , Gy Resttumör ☐ abdomen ☐ pulm ☐ annat, spec		
Kirurgi		
Datum år mån dag Orsak till kirurgi Resttumör ☐ abdomen ☐ pulm ☐ annat, Recidiv ☐ abdomen ☐ pulm ☐ annat, Annan orsak, specificera orsak och lokalisation,		
Sjukhus Patolog avd PAD nr - år		
PAD ☐ nekros/fibros ☐ seminom ☐ annat,		
Radikal operation Kirurgisk allvarlig komplikation ☐ nej ☐ ja ☐ nej ☐ ja, spec		
Fortsatt behandling efter ev. tilläggsbehandling (ny beh blankett ifylles vid behov)		
☐ ingen ☐ kirurgi ☐ högdoskemoterapi med rescue ☐ kemoterapi ☐ strålbehandling ☐ annan behandling, spec		

Not Valid

Förklaringar till Behandlingsblankett SWENOTECA VII, Seminom

1. Gradering av toxicitet (WHO 1979)

	Grad 3	Grad 4
Hematologisk (vuxna)		
Hemoglobin g/L	65–79	< 65
Vita x 10 ⁹ /L	1,0–1,9	< 1,0
Trombocyter x 10 ⁹ /L	25–49	< 25
Urinvägar		
S-Kreatinin	5–10 x N	> 10 x N
Neurotoxicitet		
Perifer	Intolerabla parestesier och/eller uttalad svaghet	Förlamning
Obstipation*	Uppspänd buk	Uppspänd buk och kräkningar
Infektion	Svår infektion	Svår infektion med blodtrycksfall

N = Övre normalgränsen

* = Obstipation, inkluderar ej obstipation p g a morfinspreparat

Not Valid

2. Remissionsbedömning. Effekt av behandling

Komplett remission:	Fullständigt försvinnande av samtliga tumörmanifestationer på CT/MR eller motsvarande. Normala tumörmarkörer.
Partiell remission:	Reduktion av mätbar tumör med $\geq 50\%$ ($\geq 50\%$ reduktion av produkten av de största perpendikulära diametrarna) utan samtidig progress på andra lokaler. HCG normalt eller faller enligt t1/2.
Stabil sjukdom:	Reduktion av mätbar tumör med $< 50\%$ ($< 50\%$ reduktion av produkten av de största perpendikulära diametrarna) utan samtidig progress på andra lokaler. HCG oförändrat eller faller enligt t1/2.
Progressiv sjukdom:	Ökning av mätbar tumör med $\geq 25\%$, eller tillkomst av nya tumörmanifestationer, eller ökning av tumörmarkörer $> 10\%$.
Ej bedömbart:	Objektiv parameter saknas (t ex vid adjuvant behandling) eller ej undersökt vid undersökningstillfället.

SWENOTECA VII, Seminom		Personnummer
Uppföljningsblankett		Namn
Svenska patienter SWENOTECA sekretariatet Onkologiskt centrum Universitetssjukhuset i Lund 221 85 LUND	Norska patienter Kontor för klinisk kreftforskning Kreftavdelningen Haukeland Universitetssykehus 5021 Bergen	
Klinik, sjukhus		
Läkare		

Besöksdatum år mån dag	
Status ☐ inga tecken på sjukdom ☐ stabil eller minskande resttumör ☐ recidiv/progress av resttumör (fyll i nedan)	
☐ kontralateral testikelcancer ☐ annan cancer	
Senreaktion av behandling år 1, 3 och 5	
Minskad libido ☐ nej ☐ ja Impotens ☐ nej ☐ ja Annan ☐ nej ☐ ja, spec.	Testosteron , SHBG LH FSH Testosteron substitution
normalt förhöjt lågt ej utfört	
Fortsatt behandling (ny beh blankett ifylles vid behov) ☐ ingen ☐ kirurgi ☐ högdoskemoterapi med rescue ☐ kemoterapi ☐ spec. behandling ☐ annan behandling, spec	

Recidiv Datum år mån dag	
Progress av resttumör ☐ nej ☐ ja	
Lymfkörtelmetastaser Inguinalt nej ja ej evaluerbart Största metastas (mm x mm) Iliakalt ☐ ☐ ☐ x Paraaortalt ☐ ☐ ☐ x Mediastinalt ☐ ☐ ☐ x Supraklav ☐ ☐ ☐ x	Extralymfatiska metastaser Lunga nej ja ej evaluerbart Största metastas (mm x mm) Lever ☐ ☐ ☐ x Hjärna ☐ ☐ ☐ x Skelett ☐ ☐ ☐ x Annat, spec
Symtom eller undersökning(ar) som först signalerar recidiv (flera alternativ kan anges) ☐ symptom ☐ CT/MR/UL ☐ tumörmarkörer ☐ annat, spec	

Dödsdatum år mån dag
Dödsorsak ☐ testikelcancer ☐ biverkningar i samband med behandling ☐ annan cancer ☐ annan orsak

Not Valid

SWENOTECA VII, SEMINOM Registreringsblankett

Svenske pasienter
Swenoteca sekretariatet
Onkologisk centrum
Universitetssjukhuset i Lund
221 85 Lund

Norske pasienter
Kontor for klinisk kreftforskning
Kreftavdelingen
Haukeland Universitetssykehus
5021 Bergen

Navn _____

Fødselsdato (d/m/å)

						1	9		
--	--	--	--	--	--	---	---	--	--

Personnr.

--	--	--	--	--	--	--	--	--	--

Sykehus, avd.

Lege

Sykehusnr.

--	--	--

Pasientnr.

--	--	--	--	--	--

Orchiektomi

☐ nei	dag	måned	år	Kontralateral testisbiopsi	Spermiogram	hvis ja	☐ før orchiektomi
☐ ja	Dato		20	☐ Cis ☐ ikke Cis ☐ ikke utført	☐ nei ☐ ja		☐ etter orchiektomi

Sykehus

Avd. Pat.

PAD nr.

										-			år
--	--	--	--	--	--	--	--	--	--	---	--	--	----

Orchiektomi	☐ hø	☐ ve	Tumorstørrelse	"Utbrent tumor"	☐ nei ☐ ja
Innvekst i rete testis	☐ nei ☐ ja			(Fylles ut bare hvis tumorstørrelse ikke er angitt)	
Vaskulær invasjon	☐ nei ☐ ja ☐ uklart				

Tumormarkører

Før orchiektomi

Dato	dag	måned	år	AFP	☐ normalt ☐ forhøyet ☐ ikke utført
			20	β-HCG	☐ normalt ☐ forhøyet ☐ ikke utført
				LD	☐ normalt ☐ forhøyet ☐ ikke utført
				PLAP	☐ normalt ☐ forhøyet ☐ ikke utført

Etter orchiektomi (ved definitiv stadieinndeling)

Dato	dag	måned	år	AFP	☐ normalt ☐ forhøyet ☐ ikke utført
			20	β-HCG	☐ normalt ☐ forhøyet ☐ ikke utført
				LD	☐ normalt ☐ forhøyet ☐ ikke utført
				PLAP	☐ normalt ☐ forhøyet ☐ ikke utført

Hormonstatus

Dato	dag	måned	år	Testosteron	☐ normalt ☐ forhøyet ☐ lavt ☐ ikke utført
			20	SHBG	☐ normalt ☐ forhøyet ☐ lavt ☐ ikke utført
				LH	☐ normalt ☐ forhøyet ☐ lavt ☐ ikke utført
				FSH	☐ normalt ☐ forhøyet ☐ lavt ☐ ikke utført

Metastaser

Lymfeknutemetastaser

Inguinalt	☐ nei ☐ ja	Største metastase (mm x mm)
Iliakalt	☐ nei ☐ ja	
Paraaortalt	☐ nei ☐ ja	
Mediastinalt	☐ nei ☐ ja	
Supraklav	☐ nei ☐ ja	

Ekstralymfatiske metastaser

Lunge	☐ nei ☐ ja	Største metastase (mm x mm)
Antall lungemetastaser		
Skjelett	☐ nei ☐ ja	
Lever	☐ nei ☐ ja	
Hjerne	☐ nei ☐ ja	
Annet	☐ nei ☐ ja	Lokalisasjon

Klinisk stadieinndeling

☐ CSI	Antall risikofaktorer ² ☐ 0 ☐ 1 ☐ 2	T-stadium pT ³ ☐ nei ☐ ja	☐ CSII ☐ CSIII ☐ CSIV ☐ CSMk+
Abdominale lymfeknuter ☐ 0 ☐ A ☐ B ☐ C ☐ D	Gruppering ifølge MRC ⁴ ☐ small ☐ large ☐ very large volume	Prognose ifølge IGCCC ⁵ ☐ god ☐ intermedieær ☐ dårlig	
Behandling CSI	☐ surveillance ☐ adj. karboplatin ☐ adj. strålebehandling		
Behandling CSII-IV, CSMk+	☐ strålebehandling ☐ EP ☐ BEP ☐ annen, spes.		

Kommentarer

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37683



1 Klinisk stadieinndeling: modifisert etter RMH

- CS I Ingen tegn til metastaser
- CS Mk+ β -HCG er vedvarende forhøyet (reduseres ikke i forhold til sin halveringstid), men ingen påvisbare metastaser
- CS II Metastaser begrenset til abdominale lymfeknuter
- A maksimal diameter <2 cm
- B maksimal diameter 2–5 cm
- C maksimal diameter >5–10 cm
- D maksimal diameter >10 cm
- CS III Metastaser i lymfeknuter over diafragma
- For abdominale lymfeknuter gjelder:
- 0 ingen metastaser
- A–D ifølge CS II ovenfor
- CS IV Ekstralymfatiske metastaser
- For lungemetastaser gjelder:
- L₁ ≤3 metastaser, ingen >2cm
- L₂ >3 – ≤20 metastaser, ingen >2cm
- L₃ ≤20 metastaser, >2cm
- L₄ >20 metastaser
- For abdominale lymfeknuter gjelder:
- 0 ingen metastaser
- A–D ifølge CS II ovenfor

2 Risikofaktorer

Primærtumor >4 cm, innvaskulære metastaser

3 pT4

Tumor invaderer scrotum, med eller uten vaskulær/lymfatiske invasjon

4 Gruppering ifølge Medical Research Council

- Small volume disease CS Mk+, II_{A–B}, III_{0–A–B}, IV_{0–A–B, L1–L2}
- Large volume disease CS II_{C–D}, III_{C–D}, IV_{C–D, L1–L2}
- Very large volume disease CS IV_{0–D, L3–L4}; ekstralymfatiske ekstrapulmonale metastaser (skjelett, lever, hjerne)

5 International Germ Cell Collaborative Consensus Classification

Good-prognosis group

All of the following criteria:

Any primary site

No non-pulmonary visceral metastases

Normal AFP

Any β -HCG

Any LDH

Intermediate-prognosis group

Any of the following criteria:

Any primary site

Non-pulmonary visceral metastases

Normal AFP

Any β -HCG

Any LDH

Poor-prognosis group

No patients classified as poor prognosis

Not Valid

SWENOTECA VII, SEMINOM Behandlingsblankett

Svenske pasienter
Swenoteca sekretariatet
Onkologisk centrum
Universitetssjukhuset i Lund
221 85 Lund

Norske pasienter
Kontor for klinisk kreftforskning
Kreftavdelingen
Haukeland Universitetssykehus
5021 Bergen

Navn _____

Fødselsdato (d/m/å)

				1	9				

Personnr.

--	--	--	--	--	--	--	--

Sykehus, avd. _____

Lege _____

Sykehusnr.

--	--	--

Pasientnr.

--	--	--	--

Årsak til behandling

- ☐ adjuvant ☐ metastatisk sykdom ☐ recidiv

Start av behandling dag måned år

		2	0

Avsluttet behandling dag måned år

		2	0

Kjemoterapi

- ☐ karboplatin ☐ EP ☐ BEP ☐ annet _____

Antall kurer

--	--

Strålebehandling

- ☐ paraaortalt ☐ paraaortalt + iliakalt ☐ annet _____

Fraksjonsdose Targetdose

--	--	--	--

 Gy

--	--	--	--

 Gy

Årsak til avsluttet behandling

- ☐ ferdig program ☐ terapivikt ☐ bivirkninger ☐ annen, spes. _____

Toxicitet ¹ Grad 3-4

Hematol. Hb ☐ nei ☐ ja
hvite ☐ nei ☐ ja
trombocytter ☐ nei ☐ ja
Renal S-kreat ☐ nei ☐ ja

Perifer neuropati ☐ nei ☐ ja
Obstipasjon ☐ nei ☐ ja
Infeksjon ☐ nei ☐ ja
Annen alvorlig toxicitet ☐ nei ☐ ja, spes. _____

Effekt av behandling ²

- ☐ adjuvant behandling ☐ C ☐ F ☐ S ☐ P ☐ Ikke evaluert

Fortsatt behandling (ruter fylles ut når slutning er ferdig og evt. tilleggshandling er gitt)

- ☐ ingen ☐ kjemoterapi, spes. ☐ høydosekjemoterapi med rescue
☐ konsoliderende strålebehandling ☐ kirurgi ☐ annen behandling, spes. _____

Konsoliderende strålebehandling (etter kjemoterapi/kirurgi)

Startdato dag måned år

		2	0

Fraksjonsdose Targetdose

--	--	--	--

 Gy

--	--	--	--

 Gy

Resttumor ☐ abdomen ☐ pulm ☐ annet _____

Kirurgi

Dato dag måned år

		2	0

Årsak til kirurgi

Resttumor ☐ abdomen ☐ pulm ☐ annet _____
Recidiv ☐ abdomen ☐ pulm ☐ annet _____

Annen årsak, spesifiser årsak og lokalisasjon _____

Sykehus _____ Avd. Pat. _____

PAD nr.

--	--	--	--	--	--	--	--

 -

--	--

 år

PAD ☐ nekrose/fibrose ☐ seminom ☐ annet _____

Radikal operasjon Kirurgisk alvorlig komplikasjon
☐ nei ☐ ja ☐ nei ☐ ja, spes. _____

Fortsatt behandling etter evt. tilleggshandling (ny Behandlingsblankett fylles ut ved behov)

- ☐ ingen ☐ kirurgi ☐ høydosekjemoterapi med rescue
☐ kjemoterapi ☐ strålebehandling ☐ annen behandling, spes. _____

Kommentarer

4809



1 Gradering av toxicitet (WHO 1979)

	Grad 3	Grad 4
Hematologisk (voksne)		
Hemoglobin g/L	65–79	< 65
Hvite x 10 ⁹ /L	1,0–1,9	< 1,0
Trombocytter x 10 ⁹ /L	25–49	< 25
Urinveier		
S-Kreatinin	5–10 x N	> 10 x N
Neurotoxicitet		
Perifer	Intolerable parestesier og/eller uttalt svakhet	Lammelse
Obstipasjon*	Utspilt buk	Utspilt buk og brekninger
Infeksjon	Alvorlig infeksjon	Alvorlig infeksjon med blodtrykksfall

N = Øvre normalgrense

* = Obstipasjon, inkluderer ikke obstipasjon p.g.a. morfinpreparat

Not Valid

2 Remisjonsvurdering. Effekter av behandling

Komplett remisjon: Samtlige tumormanifestasjoner er fullstendig forsvunnet på CT/MR eller tilsvarende. Normale tumormarkører.

Partiell remisjon: Reduksjon av målbar tumor med $\geq 50\%$ ($\geq 50\%$ reduksjon av produktet av de største perpendikulære diametre) uten samtidig progresjon i andre lokalisasjoner. HCG normal eller er redusert likt t1/2.

Stabil sykdom: Reduksjon av målbar tumor med $< 50\%$ ($< 50\%$ reduksjon av produktet av de største perpendikulære diametre) uten samtidig progresjon i andre lokalisasjoner. HCG uforandret eller er redusert likt t1/2.

Progressiv sykdom: Økning av målbar tumor med $\geq 25\%$, eller tilsynekomst av nye tumormanifestasjoner, eller økning av tumormarkører $> 10\%$.

Ikke evaluerbart: Objektiv parameter savnes (f.eks. ved adjuvant behandling) eller ikke undersøkt ved kontrolltidspunktet.

SWENOTECA VII, SEMINOM Oppfølgingsblankett

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221 85 Lund

Norske pasienter

Kontor for klinisk kreftforskning
Kreftavdelingen
Haukeland Universitetssykehus
5021 Bergen

Navn

Fødselsdato (d/m/å)

Personnr.

Sykehus, avd.

Lege

Sykehusnr.

Pasientnr.

Besøksdato

dag måned år
. . 2 0

STATUS

☐ ingen tegn til sykdom (NED)

☐ stabil eller minskende resttumor

☐ recidiv/progresjon av resttumor (fyll ut nedenfor)

☐ kontralateral testikkel cancer

☐ annen cancer

Senreaksjon av behandling år 1, 3 og 5

Minsket libido

☐ nei ☐ ja

Impotens

☐ nei ☐ ja

Annen

☐ nei ☐ ja, spes.

Testosteron

. ,

normalt forhøyet lavt ikke utført

SHBG

LH

FSH

Testosteron substitusjon

☐ nei ☐ ja

Fortsatt behandling (ny Behandlingsblankett fylles ut ved behov)

☐ ingen

☐ kjemoterapi

☐ kirurgi

☐ strålebehandling

☐ høydosert kjemoterapi med resttumor

☐ annen behandling, spes.

Recidiv

Dato

dag måned år
. . 2 0

Progresjon av resttumor

☐ nei ☐ ja

Lymfeknutemetastaser

	nei	ja	ikke evaluerbart	Største metastase (mm x mm)
Inguinalt	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Iliakalt	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Paraaortalt	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Mediastinalt	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Supraklav	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐

Ekstralymfatiske metastaser

	nei	ja	ikke evaluerbart	Største metastase (mm x mm)
Lunge	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Lever	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Hjerne	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐
Skjelett	☐	☐	☐	☐ ☐ ☐ X ☐ ☐ ☐

Annet, spes.

Symptom eller undersøkelse(r) som først signaliserte recidiv (flere alternativ kan angis)

☐ symptom ☐ CT/MR/UL ☐ tumormarkører ☐ annet, spes.

Dødsdato

dag måned år
. . 2 0

Dødsårsak

☐ testikkel cancer

☐ bivirkninger i forbindelse med behandling

☐ annen cancer

☐ annen årsak

Kommentarer

57677